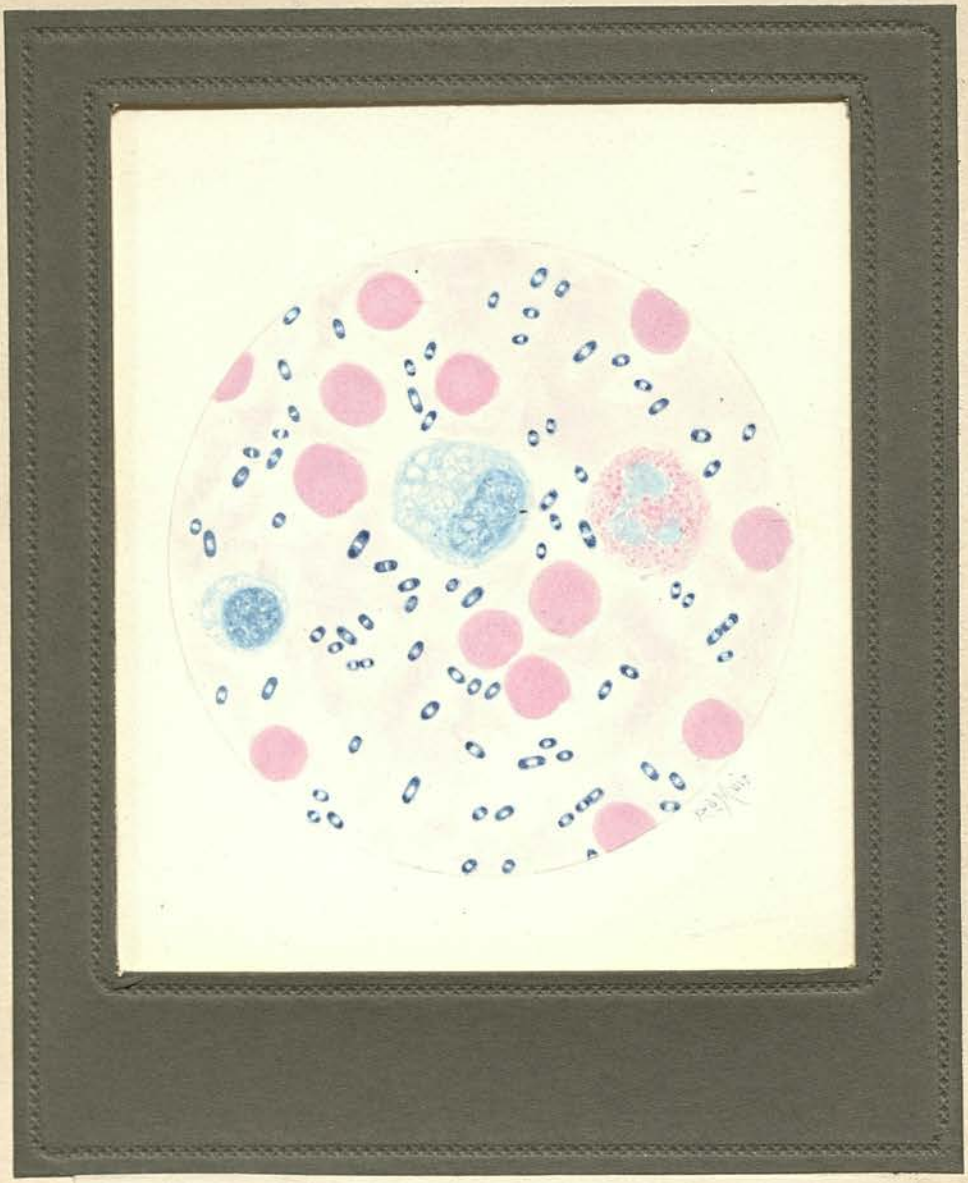
# PLAGUE

A THESIS FOR THE DEGREE OF M.D.

by

J. C. CARR, M.B., Ch.B. 1899.



Bacillus Pestis. Coloured Micro-drawing.
Film preparation.- Scraping from spleen
of guinea pig. Experimental plague.
stained with methylene blue and eosin.

In justification of my selection of the subject of plague for a Thesis I will say at the outset that it appeared a suitable one as I have had some practical experience during the cool season of 1902-3 in the Bombay Presidency of India, of the disease, with regard to its symptoms, bacteriology and treatment, both prophylactic, and directed towards its favourable termination when already set in. Apart from curative serums such as that prepared by M.Yersin, of which I have had no practical experience, there is no known agent which will destroy the organism of plague when once it has penetrated into the system, and thus treatment is of necessity general, rather than specific.

During four months, from the latter end of July to the close of November, I was assisting in the work of the Miraj Medical Mission and Medical School, at the invitation of Dr. J. Rutter Williamson. Miraj is a Hindu town of some 25.000 inhabitants - situated in the Southern Mahratta County, a native state, and 150 miles along the Southern Mahratta Railway and the South of Poona. The school is carried on under the auspices of the American Presbyterian Missionary Society, and is largely indebted for its present flourishing condition to the energy and determination of the Principal, Dr./

Dr. W.J.Wanless, who started the work on quite a small scale some thirteen years ago. The establishment being financed by a generous friend in Canada, Dr. Wanless has had a free hand with regard to furnishing the hospital with many of the newest applicances, such as high pressure hot air sterilising apparatus; and operations are now performed in great numbers week by week, under as favourable circumstances as exist in the most modern establishments in England or Scotland.

Miraj was attached by Plague in the year 1897, the same year which witnessed such a fearful mortality in the adjoining town of Poona. It was infected, along with Poona and other towns on the route to Belgaum, Hubli and the south, from the centre of Bombay, where plague broke out in 1896. Each year since 1897 it has occurred in greater or less severity in the town of Miraj, the onset being declared at the close of the rainy season or thereabouts, and the decline in the early months of the following year, January and February - its greatest severity being during the coolest season of the year.

In writing this thesis I have consulted the writings of various authorities on the subject.

Among these being Bede, Gibbon, Creighton, Scheube, Cantlie, Netter, Jennings, Simond, Clemow, Hankin/

Hankin, Manson, Batzaroff, Surveyor, Childe, Muir and Ritchie, Balfour Stewart, Kitasato and Nakagawa.

I have consulted largely the Blue Book Report of the Indian Plague Commission.

My thanks are due to Alexander Wilson M.D., of the American Presbyterian Mission and Medical School, Miraj, Southern Mahratta County, India, for kindly furnishing copies of plague charts; to Dr. J. Rutter Williamson M.D., Edin. for information. concerning his own case; to Dr. J. W. Ballantyne and Dr. R. A. Fleming, for permission to consult books in the Library of the Royal College of Physicians, Edinburgh; to Dr. Theodore Shennan for an introduction to the Library of the Royal College of Surgeons, Edinburgh. To Mr. Richard Muir I am indebted for four photographs and a drawing of the Plague Bacillus.

In all probability the disease now known as Bubonic plague, Plague, Peste or Malignant Poly-adentitis,* occurred in very ancient times.

In Biblical history[1] we find an account of a scourge which destroyed the Philistines in Ashod whilst they were in possession of the Ark. The "emerods in their secret parts" referred to in this account possibly were identical with the swellings now known as inguinal buboes. The Israelites in Dueteronomy 28 verse 27 are threatened, "with the botch of Egypt, and with the Emerods, and with the scab, and with the itch, whereof thou canst not be healed."

It is interesting to notice in this account the association of mice with the "deadly destruction" due to this plague. There is no mention of mortality occurring among these animals although they were referred to as marring the land[2], but some connection must have been supposed between mice and the emerods, otherwise they would scarcely have been associated together in the trespass offering[3] offered by the Philistines on the recommendation of their priests and diviners.

Another fact worthy of mention is that although no person accompanied the ark to Bethshemesh, the plague attacked the people of that place.[4]

Josephus[5] throws further light upon this outbreak. He/

* James Cantlie, M.B., F.R.C.S., Eng. Address before before the Epidermological Society. British Medical journal. Jan 9 1897 p.72

1. 1 Samuel V. 6.9
2. 1 Samuel VI. 5
3. 1 Samuel VI. 4
4. 1 Samuel VI. verse 19.
5. Antiquities of the Jews. Book VI. Chapt.1. p.347.

He tells of rapid death caused by "dysentery," accompanied by the vomiting up "Entrails," eaten through and rotted away, and also of a scourge of mice, which came to the surface from their holes, and devoured fruit and vegetation.

Our knowledge however, of these early and fearful epidemics is very scanty and we can only surmise as to whether they were genuine plague, or some other fatal disease. Authors differ in their opinions. Manson holds that the descriptions are not accurate enough to justify a positive opinion.[1] Scheube holds the same view.[2] But it is to the records of more recent times that we must turn in order to obtain really authentic accounts.

Daremberg shows that plague existed two centuries before the Christian era. Interesting knowledge has been gathered from Oribasius' Manuscript[4] which was found among the palimpsests by cardinal Mai. In this manuscript there is an account by Rufus of Ephusus (98-117 A.D.) of the plague that occurred in his time in Egypt, Lybia and Syria. He says that buboes occurred in those attacked, and he suggests that the disease is identical with the 'maladie a bubons' of Hippocrates, also stating that those living near marshy places seemed specially liable to attack/

1. "Tropical Diseases" page 231.
2. "Disease of Warm Climates." p.5
3.
4. M. le Dr. Netter.
La Semaine Medicale. 1895. p.60

attack.

Dioscoride and Posidonius in their treatise on the plague in Lybia in their time describe fever, delirium and buboes.

A plague occurred under the Emperor Marcus Aurelius, which seemed to follow Lucius Verrus from province to province of the Empire. Gabin says that it presented symptoms like these described by Thucydides as occurring in the plague of Athens in 430 A.D. The latter writer describes the symptoms observed with great accuracy, referring to the nervous, respiratory, and digestive symptoms, gangrenes and eruptions. If he did not refer to the occurrence of buboes, Netter observes, it is because they did no exist, and so neither this plague nor that in the time of Marcus Aurelius can have been "bubonic" in character, although this, as we shall see later, does not necessarily imply that they were not genuine plague.

The next account we can rely upon as being authentic is that of the great plague which occurred in the sixth century in the time of the Emperor Justinian, who ruled from 527 - 565 A.D. It is described by Evagrius and Procope, and was evidently/

evidently bubonic plague. It not only infected the shores of the Mediterranean and Persia, but spread over almost the whole of Europe from country to country - and lasted for between fifty and sixty years, the disease remaining in Rome and elsewhere during the following centuries. The mortality was fearful, towns being depopulated, and terrible distress prevailing. Gibbon [1] goes into considerable detail in this description of the disease and its ravages. He calls it the "fever of the earth," thus adhering to the ancient theory that the disease rose up from the ground. He considers that Egypt and Ethiopia were the source of the disease, on account of their hot, damp, stagnating atmosphere, impregnated with the products of putrefaction of animal and vegetable matter, and especially mentioning the swarms of locusts. It first appeared in Pelusium between the Serbonianbog and the Eastern channel of the Nile - spreading from there in two directions: to the East over Syria and Persia; to the West, along the coast of Africa, and over the continent of Europe. In the spring of the second year it attacked Constantinople, and is described by Procopius, who observed that in some cases the attack was preceded by delirium; in other by slight fever/

1. Gibbon. "Roman Empire." Milman Vol.V. pp.253-256

fever, followed in a couple of days by swelling of the glands, especially of the groin and axillae, and under the ear. These swellings in being opened were found to contain a black substance.

If these buboes suppurated, the patient was often saved, if not, death generally ensued on the fifth day. Lethargy or delirium often accompanied the fever, and the body was 'covered with black pustules or carbuncles, the symptoms of immediate death.' The plague was generally fatal in the case of pregnant women; but an instance is recorded of a child being extracted alive from its dead mother, and of three mothers surviving the loss of dead foeti.

All ranks were attacked, males more than females and aphonia is described as a frequent sequela. No drug was of any certain efficacy. Justinian himself was attacked by the disease, but recovered. Procopius believes in the contagiousness of the disease by respiration into the lungs and stomach, from sick to healthy persons, and thinks that the popular disbelief in this power of contagion may have helped towards its rapid spread. No system of quarantine, and no restraint of intercourse was insisted on between province and province- the people were mingled by war and emigration - and infected/

infected merchandise passed from place to place.

Procopius remarks that it always spreads from the coast inland, and that the air was infected to such an extent that seasons had no effect on it, although he believed that it would soon die out in cold and temperate climates. During three months from five to ten thousand persons are said to have died in Constantinople each day.[1] Many cities in the East were depopulated and deserted.

The above is a brief summary of Gibbon's account in which we see an almost exact analogy of the bubonic plague in the East at the present time.

I will just emphasise the main points of similarity.

(1) Origin in a recognised centre.

(2) Spread of infection along trade routes, from the sea coast inland.

(3) Mode of onset of the disease in the individual.

(4) The appearance of buboes after a period of invasion, and the situation of these buboes.

(5) Resolution of the attack following upon suppuration in the bubo.

(6) Death in fatal cases occurring commonly on the fifth day. As regards this point, however,/

1. Gibbon. Roman Empire. Milman. Vol.V. pp.253-256.

however, modern authorities gather from statistics of plague epidermics that death is usual from the third and the fifth day, the liability to death decreasing as the third day is passed.

(7) Lethargy or delirium accompanying the fever.

(8) Mortality great amongst pregnant women.

(9) Youth most liable to attack.

(10) Aphonia a common sequela.

(11) No specific drug cure as far as it is known.

(12) The disease was not fatal to the Emperor Justinian as now the mortality is less amongst the higher classes than amongst the lower.

(13) Contagiousness.

(14) Infection contained in merchandise, in this case a bale of cotton.

(15) Alternate languishing and reviving of the epidermic.

The points of difference may also be noted —

(1) Presence of black pustules or carbuncles not common in modern epidermics.

(2) Vomiting of blood, which is not a frequent symptom.

(3) All ranks were attacked with indiscriminate rage/

rage.

(4) No mention is made of any mortality amongst rats, or any other of the lower animals.

One hundred years later the plague appeared in England. An account of it is to be found in Bedes Ecclesiastical History.[1] Bede is the first contemporary or almost contemporary authority. At this time there was immense mortality in the South and in Northumbria. Many notables died in Ireland, and one vaguely estimates that one third of the population of that island were left alive after the scourge.[2] It is curious to relate that in the year of this plague, 664 A.D., there was an eclipse of the sun. Tuda, Bishop of Northumbria, was carried off by it. Aethelhun and Ecgberht, young English Nobles of much ability, were attacked in Ireland in the Monastery of Rathmelsigi, now Mellifont. Aethelhun succumbed but the story runs his companion, giving himself to prayer and other devotional exercises, recovered.

At this time many monasteries were attacked, with great mortality amongst the monks and nuns.

A/

1. Bede's Ecclesiastical History. Part I Section 2 p.241.

2. Creighton. "A history of Epidemics in Britain." p.5.

A notable example is found in the case of one of the monasteries at Jarrow. One story relates of many deaths occurring on the male side of the house.

Few of the highest classes died of the disease, but a great number of clergy and poor people. The deaths in London are variously estimated at between twenty to one hundred thousand.

But probably the greatest epidemic on record is that known as the Black Death, of the fourteenth century. This, as Pagel says "as regards social and political conditions, even of the most intimate family ties, and it had as a consequence, in the truest sense of the word, a deterioration of all social and friendly relations." The whole of the then known globe was smitten and depopulated by it. Hecker estimates that twenty five millions of persons fell victims to it in Europe alone.

Pope Clement VI gave authority for a report to be published giving the mortality as 42,836,486 out of a population of 105,000,000! In the writings of Cantacazena, Guy de Chauliac, Chalin de Venario, descriptions of tumefaction and ganglionic suppuration occur. Littré published in the library/

1. Creighton.
2. La semaine Medicale. 1895 p. bg. M.la Dr. Netter.

library of the Ecole de Chates a manuscript composed in 1350 by Symon of Corius, in which it is described as'pestis inguinaria.' He indicates the early appearance of swellings, and the great contagiousness of the disease. At this time also disturbances were noticed in the heavens, such as comets, and the conjunction of Saturn, Mars and Jupiter. The Jews suffered great persecution, being accused of poisoning the wells with a mixture of spiders, blood from buboes, and poisonous animals.

'You enter in your Florence wanderings,
'The Church of Saint Maria Novella, pass
'The left stair, where at plague-time Machiavel
'Saw One with set fair face as in a glass,
'Dressed out against the fear of death and hell,
'Rustling her silks in pauses of the mass,
'To keep the thought of how her husband fell,
'When she left home, stark dead across her feet.[1]

Bocaccio was an eye witness of the disease in Florence - where he says that it commenced with "bleeding of the nose, a sure sign of inevitable death." Animals fell sick and shortly expired if they but touched articles belonging to the sick or dead. He relates having seen two hogs tearing at/

1. Mrs Browning. Casa Guidi Windows. Part I lines 320-327.

at the clothes of a patient recently dead of the plague; shortly afterwards they fell down and died, after staggering about for a time. In other places cats, dogs, fowls and other animals were attacked.[1]

Cleyn describes haemorrhage from the lungs as occurring at this time - and this is distinctive of the Black Death among other bubonic plague epidemics. He mentions death from "carbuncles and boils and botches which grew on the legs or under the arms; others died from passion of the head, as if thrown into a frenzy; others by vomiting blood."

Woodhall in 1637 writes " But the pertilential bubo or boyle cometh ever furiously on, and as in a rage of feaver, and as being in haste; sometimes it lighteth in or near the inguen thwart, but more often upon the thigh, pointing downward with one end, the uppen end towards the belly being commonly the biggest or the fullest part of the bubo, the whole thigh being also inflamed.

"A treatise faithfully and plainly declaring the way of preventing, preserving from, and curing that most fearful and contagious disease called the Plague, with the Pestilential Feaver and other the fearful symptoms and accidents incident thereto."

1. Jennings "A manual of Plague" p.7

By John Woodhall,
Surgeon to St. Bartholomew's Hospital
LONDON. 1639.

In the 1665 occurred the Great Plague of London I do not propose to give a lengthened account of this epidemic, the mortality caused by which was in one week, that ending September 19th 1665, estimated, and probably underestimated, at 10,000 A description, quaint and old fashioned, but very vivid, is given by Defoe.[1]
The type of plague was not bubonic, and Manson does not even mention it in his sketch of the history of plague. It is however, considered to have been of that nature by some.[2]

Defoe speaks (under "Orders concering infected houses and persons sick of the Plague") of "botch, or purple or swelling in any part" of the body, we read (pp.91 - 92) that cats, dogs, rats and mice, were ordered to be destroyed; a great number of the latter were destroyed. No mention is made however, of spontaneous mortality amongst these animals.

In/

1. Defoe. Vol. V
2. Report of Indian Plague Commission p.52.

In more recent times the "Oriental Plague" occurred in Turkey, Egypt and the Levant, in the early part of the nineteenth century, also the plague in Western India from 1812-1837.
Both these were genuine bubonic plague.[1]

It may be well to consider shortly the ancient theories held regarding the etiology and spread of the disease. These theories were various.

Etienne Pariset believed that in Egypt it was spread from rapidly putrefying dead bodies. He extolled the practice of embalming as carried out in ancient Egypt: and argued that it was due to this that the country enjoyed comparative immunity from the disease. This embalming ensured slow mouldering of animal bodies, beyond the reach of the yearly inundations of the Nile. True it is that there had been epidemics in Egypt, but they were of typhus, and other sicknesses of slavery, prisons and famines. Herodotus says that Egypt and Lybia were the ~~less~~ healthiest countries under the sun. With the advent of Christian doctrine came also the practice of burial of the dead. Plague had indeed occurred previous to this, but embalming had been far from the general custom. For the poorer people there were less/

1. Report of the Indian Plague Commission p.53

less expensive methods of embalming, ensuring slow mouldering of the remains.

It was also thought that the yearly inundation of the Nile affected the salubriousness of the country injuriously, soaking the land with the unhealthy products of decomposition, with risk to those living upon it.

It would serve no purpose to go further into the theories held. They were based on supposition, and on a certain amount of practical experience, but were without that foundation of Scientific knowledge which alone can firmly establish any theory.

ENDEMIC CENTRES OF PLAGUE.

There are certain areas on the surface of the earth where plague appears to be endemic; that is to say, where the virus exists, and is kept alive by some means, without being introduced from any outside source. The study of these endemic centres is one of great interest. In them, plague is continually smouldering, at times increasing in virulence and spreading to the districts lying around, then again subsiding, perhaps formany years, before becoming again virulent.

One of these centres and perhaps the most important centre of all, is situated in South East China, in the province of Yun-nan.

Rocher/

Rocher, an official in the service of the French Imperial Maritime Customs of China, writes of this centre in 1878. He thinks that it was probably introduced into the province of Yun - nan from Tibet, and that these it has developed in the valleys with the town of Mengtsz as its centre . Michoud goes further, and gives it as his opinion that the plague may have come from India, through Tibet, carried along the trade routes at the time of the Mohammedan rebellion in 1859. Thus the Yunnanese "Yang-tzu-ping "would be identical with the "Mahà-mari" of India.

Since this time it has been endemic in this town of Mengtsz. A lady and gentleman who resided there from June 1896 to November 1899 state that an epidemic appearsnce annually in the town, lasting during the rainy season from June to August, and that there is an annual mortality in Mengtsz and the surrounding places of 300 -1400 from it. Rats are very abundant in the town and it is a familiar sight to see them dying of plague. Mengtsz is pathetically called the "City of Widows."

Clemow, writing in the Journal of Tropical Medicine/

1. La province chinois de Yunnan. Emile Rocher, de l'administration des Donanes Imperiales deChine. Notes sur la peate de Yunnan. Paris 1878
Buboes Parliamentary Papers Chini No.3 (1878) pp.22,23. Quoted by Clemow.

# CHINA AND JAPAN

SCALES TO CHINA

Natural Scale 1:6,198,300=97·8 miles to an inch.

Geographical Miles, 60=1 degree

Chinese Li, 192·86=1 degree

French Kilometres, 111·3=1 degree

English Statute Miles, 69·16=1 degree

*Treaty Ports underlined thus* Shanghai

*Submarine Telegraph Lines thus* S.T. or Sub. Tel.

*Railways* ———

1894

→ - - - = direction of spread of plague.

To India

Heights in English Feet. Sites of Battles &c 1856-8.

Longitude East of Greenwich.

Tropic of Cancer

30 28 26 24 22 20 18

100 102 104 106 108 110 112 114 116 118 120

Medicine. March 1900 page 201, states that a Chinese writer in Nature, Feb.16 1899 ( M.Kumagusa Minakaba) tells of its long existence in Yunnan.

Lowson states that the first known epidemic in China was in 1844.

(Indian Medical Gasette. Jan. 1897.)

This centre was vert active in the years 1871 - 73, after the Mohammedan rebellion.[1]

The Plague first appeared in Pakhoi in 1867[2] and each year from 1871-77 is was epidemic. It appeared again in 1882. There was none in 1883 but very severe again in 1884. From 1884 to 1894 there was no plague, but during the latter year it was very severe.[3]

This fact as pointed out by Clemow, is of great interest, because at this time it spread far and wide and was especially active at the endemic centre. Commercial intercourse between Yunnan and Kwang Si is incessant. Other epidemics did not show special activity at this time, so its spread over the world probably took place from China.

From this centre it spread to Pakhoi, on the Gulf of Tonking, following the Trade route by Lieun Tcheou. From the latter place its course lay down /

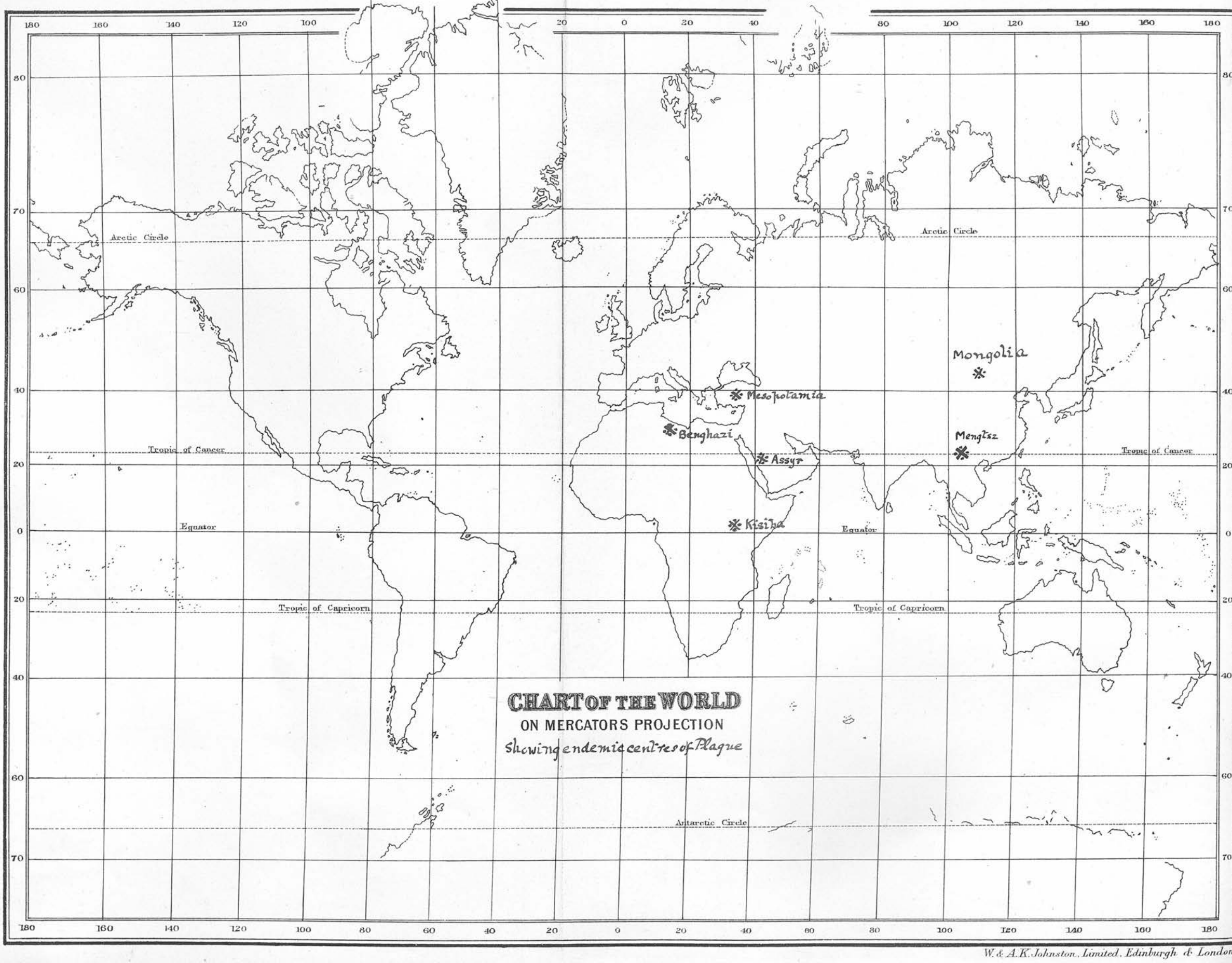
1. Manson. P.233. "Tropical Diseases."
2. Journ. Trop. Med. 1900 p.203 Clemow.
3. Sharpe Dean. Medical Reports No.47

down the caravan track along the Zung River to Canton, where it broke out with great severity in 1894. From Canton it spread to Macao in the West and to Swatow and Amoy by Sea in the North East, and on to Foochow, Formosa, and other places in South East China.

From China and Japan, by way of the trade routes it has spread to nearly all parts of the world.

In 1896 it appeared in Bombay. There has been considerable discussion as to how it was introduced there. The two possible sources are, firstly, the Himalayas, the Southern Slopes of which were probably infected with the Maha mari from Southern Tibet, and China.

There is little than can be said in favour of the Himalayan origin. Intercourse between that region and Bombay is slight, whilst between Hong-Kong and Bombay by sea it is constant. Then again at that time there was no increased virulence of plague south of the Himalayas, whilst at Hong-Kong and in South East China generally it was exceedingly virulent. The probability is, therefore, that Bombay was infected from the more virulent centre, and from that with which it was in constant commercial communication. The exact way in which the infection was carried is not easy to determine, but it may have been carried in some Chinese crackers/




# CHART OF THE WORLD

## ON MERCATORS PROJECTION

Showing endemic centres of Plague

W. & A. K. Johnston, Limited, Edinburgh & London.

crackers imported from Hong Kong.[1]

If not in this way probably by infected rats.

From Hong Kong and Japan by way of the trade routes, it has spread to practically all parts of the world, including the continent of America, where previously it had never been seen.[2] In 1899 it appeared in Glasgow.

A second endemic centre exists in Mesopotamia, where many epidemics have occurred, the last appearing in 1892.[3] From this centre the epidemics in Persia may be traced.[4]

In the Western mountainous districts of Arabia there is a centre at Assyr.[5]

In Africa there are two regions where plague is endemic. One lying in the North, Benghazi. Plague appeared here in an Arab encampment, and has broken out several times since. There is nothing to show that it was imported into the district from outside. The other African centre has been studied by Koch. It lies in Uganda. Koch, in co-operation /

1. Report of the Indian Plague Commission. Dr.Simond Annalesde l'Institut Pasteur. Oct 1898.
2. Manson. p.233. "Tropical Diseases."
3. Manson. p.232. Scheube p.5. "Diseases of warm Climates."
4. Scheube. p.5
5. Scheube. p.5. Clemow. Journal of Trop. Med. 1900 p.227.

co-operations with Zupitza established bacteriologically[1] the fact that a disease occurring in Kisiba, in the extreme North West of German East Africa, called by the natives "Rubwunga," is identical with plague. The disease is most severe in Buddu. Scheube considers[2] that without doubt this is the source of the epidemics that have occurred in Egypt from time to time, carried thither by importation of slaves. He also notices that in time this Uganda centre may attain great importance through the railway, now completed, between that country and the coast.

There has been difference of opinion among those comeptent to judge as to how the disease became imported into Uganda. Koch[3] was of opinion that it came from the north, along the line of route from Egypt, whilst Bishop Hanlon maintained that it had its origin in the south, and came from German East Africa by the route along which travelled Speke, Grant, Stanley, the first Uganda Missionaries, and the French Fathers.

We/

1. Clemow. Journal of Tropical Medicine. Feb 1900 p.192
2. "Diseases of Warm Climates." p. 5
3. Koch.Hygenischa Rundschan. Berlin. July 15. 1898.
4. The Right Reverend Bishop Hanlon. "The plague in Uganda." Manchester Memoirs. Vol. XLIII. (1899) No.6

We are indebted to Clemow [1] for a valuable article in which he describes the occurrence of a disease in Mongolia resembling Bubonic Plague in many respects, associated with a disease among a species of Marmot, the Tarmagan (Arctomys Bobac) The inhabitants hunt this animal for the sake of its fat, which they use for dressing harness and which is said to be a specific for rheumatism. These Tarmagans hibernate during the winter from September to March. If they do not go into their holes as early as they should it is a sequal to the natives that they are being attacked by a disease which they call plague (Tchuma ). The animal becomes languid, its gait unsteady, it tumbles about, cannot find its way to its hole,and becomes the prey of any enemy it may encounter. If it finds its hole the others may prevent its entrance, or if it has got in they will carry it out again. Sometimes swellings are found in these animals, under the shoulder, tense and reddish. The disease in man appears to occur solely from contact with these diseased Tarmagans. The people in these out of the way districts live in an extremely insanitary and filthy way, thus favouring the spread of any disease.

So/

1. Journal of Tropical Medicine. Feb. 1900.

So far there is no bacteriological or pathological evidence to prove that this disease in man is identical with Bubonic Plague. A doctor and a hospital assistant were sent by the Russian Government to make autopsies on the bodies of six Buriats who had died of the disease. They both were attacked and died, and Dr. Rieshetnikof, who saw them, describes in both a red and painful glandular swelling in one axilla, in which suppuration did not occur.

The disease which has been endemic in the Southern Slopes of the Himalayas called 'Maha-mari,'[1] or the Great Death is also to be identified with plague. It has been reported from this district from 1823 onwards.[2]

Evidence was obtained by the Indian Plague Commission making it quite clear that the disease was not introduced into the Kumaon and Garhwal districts from Tibet. Captain Walker and Lieutenant Doyle went to the foot of the passes leading over into Tibet, meeting certain Tibetan envoys, who year by year, according to custom, to satisfy themselves before/

1. "Maha-mari," on the Plague, in British Garhwal and Kumaon." N.G.Hutcheson, M.D., Brigade Lieutenant Colonel. Indian Medical Service.
2. Transactions of the First Medical Congress. 1894.

before the opening of the trade routes, that there is no epidemic disease in British territory. They asserted that no disease such as plague was known in Tibet. [1]

Epidemics in India have occurred from time to time, both before[2] and during the nineteenth century.[3]

In 1399 an epidemic occurred in the part ravaged by the Mursalman Timur.

In 1443 amongst the army of Sultan Ahmed I.

In 1590-1594, a famine was followed by pestilence.

In 1718 there was a famine and plague.

From 1812-1821 there was plague in Cutch, Gujerat, and Sind.

In 1824 in Kumaon and Garhwal on the Southern Slopes of the Himalayas - also in the same districts from 1834 - 1837, 1846-1853, 1876 and 1884.

In 1829 in Delhi.

In 1836 in Pali in Rajputana, at Bareilly, and Rohilkund, and spread to Jodhpore and Marwar. This epidemic lasted till 1838.

In 1896 it appeared in Bombay, and at Calcutta and spread over the greater part of India.

Before/

1. Rep. of Ind. Pl. Comm. Ch.III p.175.
2. Hankin. Annales de l'Institut Pasteur 1897 p.170.
3. Manson. p.232. "Tropical Diseases."

Before proceeding to the Bacteriology and Epidemiology of Plague it may be well to notice references made prior to the year 1894 in which year the Bacillus was discovered, to the behaviour of certain of the lower animals, in connection with Plague epidemics. Historical references are numerous. The connection noted above between the plague associated with emerods and mice which devoured the land, is of interest.

In the Bhagavati Puran, one of the Purans or religious Hindu books, instruction is given to leave the place as soon as an epidemic is observed among the rats, when rats "fall from the roof above, jump about and die." This order undoubtedly has reference to plague.

It has long been observed that previous to an outbreak of plague in any given place, a great mortality has been observed among the rats in that place.

[1]Rocher in 1878, writes of the plague in Yunnan, "Rats are first affected and as soon as they sicken they leave their holes in troops, and after staggering about, and falling over each other, drop down dead.

The approach of bubo plague may often be known from the extraordinary behaviour of the rats, who leave their holes and crevices, and issue on to the floors /

1. Reference given page 19.

floors without a trace of their accustomed timidity, springing continually upwards from their hind legs as if they were trying to jump out of something. The rats fall dead, and then comes the turn of the poultry, pigs, sheep and oxen.

"In the autumn of 1881, on the opposite side of the Yang-tsi from Nanking, and in the Western suburbs of the ancient capital, the rats emerged from holes in dwellings, jumped up, turned round, and fell down dead. Baskets and boxes filled with their bodies were cast into the canal.

Rennie says that the Chinese in Canton recognised in great mortality amongst rats a sure prognostication of the outbreak of plague. Thet would come out of their holes in broad daylight, stagger about as if dazed, and die. In a very short time one Chinese officer collected upwards of 22,000 dead rats.

Pringle says that in Shurwal in the Himalayas, where plague was epidemic in 1864 there was an exodus of rats from the villages previous to the advent of the disease and that the natives seeing this fled also.[1]

Sablonowski remarked in 1884[2] that during the epidemic in Mesopotamia of that year a certain species of fly appeared and disappeared with the outbreaks of plague.

Yersin/

1. Manson. p.242. "Tropical Diseases."
2. Manson. p.241. " "

Yersin in 1894 found that flies died in great numbers in his laboratory in Hong Kong, and that their bodies were crowded with plague bacilli. He injected a trituration of these in bouillon into a guinea pig,[1] which died soon after with all the symptoms of plague.

Since Yersin's and Kitasato's discovery in 1894 various observations have been made with regard to the incidence of plague among the lower animals - monkeys,[1] guineapigs,[2] squirrels,[1] birds,[3] mice,[1] serpents,[4] jackals,[4] sheep,[5] and oxen [6] are all mentioned as being susceptible to plague and inoculation experiments have been successfully performed upon them, inducing attacks of plague. Cats [7] have been observed to suffer from the disease.

Reference had already been made (p.24) to the connection between the disease in the tarbagan to plague in man, as pointed out by Clemow.

No herbivoro are attacked in nature.[4]

Bacteriology.

1. M.le Dr.Simond L'annals de l'Institut Pasteur.Oct 1898.
2. M.le Dr.Batzaroff L'annals de l'Institut Pasteur May 1899 p.392.
3. Dr.Surveyor, Bombay British Med.Jour. Dec.12 1896 p.1739.
4. Cantlie, Brit. Med. Journal. Jan.9. 1897 p.72
5. Rocher v. p.19.
6. " " " "
7. H.Lorans M.D., D.P.H. Ed. Brit. Med. Jour. Sept 16. 1898.
   James Cantlie, M.B., Aberd. F.R.C.S. Eng. address Epidemiological Society. Brit.Med. Jour. Jan.9. 1897 p.72

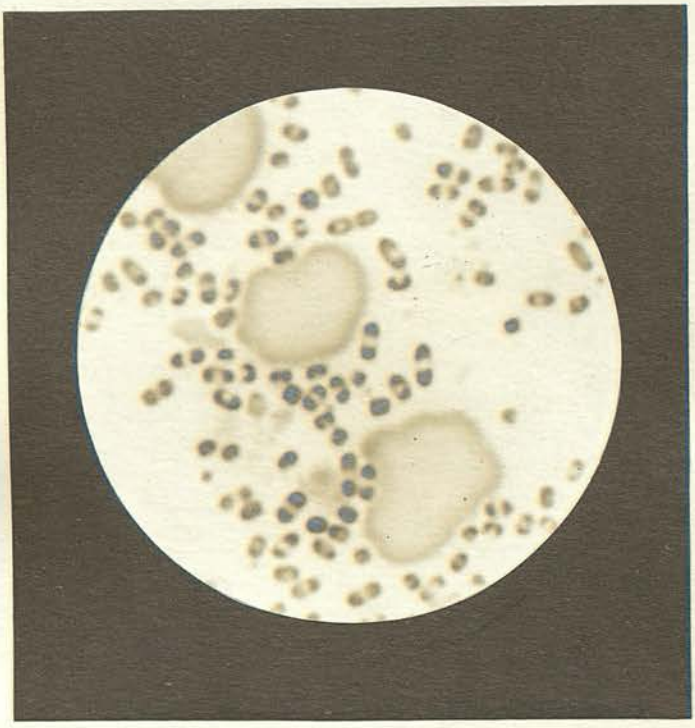
Bacteriology.

Previous to the year 1894 the Bacillus of Plague was unknown.

True it is that it was pretended by Athanae Kircher,[1] a Jesuit, that he had found in the pus of the buboes animalcules which were the germs of the disease - at the close of the 17th century. Also that in 1889 the French Government charged M.Pasteur with the inauguration of a programme of research in order to establish the microbe of the disease; but unfortunately on account of the political situation prevailing at the time, the mission which was to have been to Assyr, was not entered upon. At this time also M. Mahé suggested that the fact of the great liability to plague presented by various animals such as rats and mice might be of assistance in researches directed towards discovery of the germs. So was the honour of the discovery reserved for five years and it fell to the lot of a Japanese, Kitasato, who was sent to Hong Kong by the Japanese Government in 1894, to carry on research work there during the great epidemic in that year. This honour was shared by a Frenchman, Yersin, who was given a similar commission to the same errand by the French Government.

The/

1. M. le Dr. Netter. La semaine Medicale 1895 p.99.



Bacillus Pestis. 1 x 2000

Scraping from a lymphatic gland.

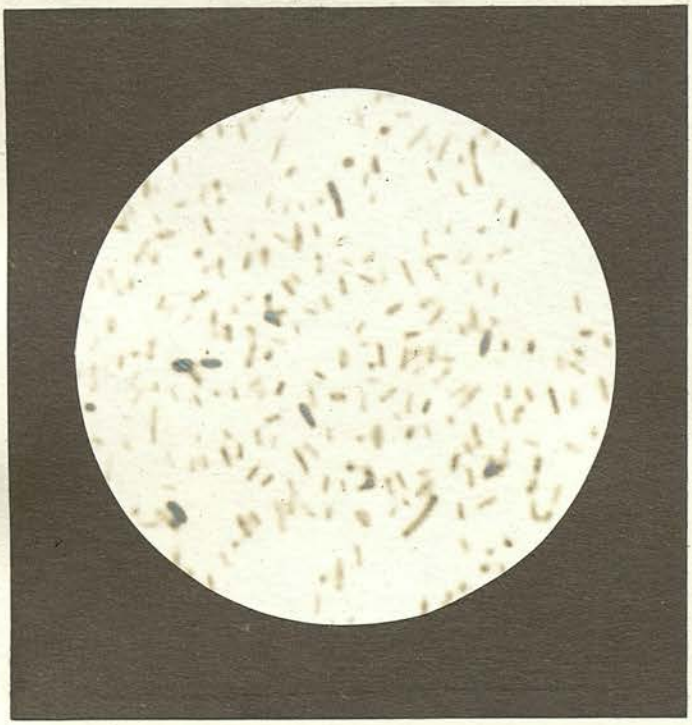
The experiments by which they almost simultaneously evolved the same results were very similar, and consisted in studying the contents of the bubo microscopically , both directly and aftercultivation on artificial media: by inoculating animals with cultures; producing plague in them, and finding post mortem the same micro-organism in the organ and blood of the animals inoculated.

Yersin found in the contents of the buboes great numbers of small rodlets, their long diameter being scarcely greater than their broad, and their poles colouring more deeply than their centres. It appeared often as if there were two cocci, separated by a clear space. These bacilli were also obtained from the bodies of the rats and mice that were found dead on the outbreaks of the disease.

Drs. F. Childe and N. F. Surveyor found bacilli in a dead rat in Bombay in 1896. Fluid from the spleen was injected into a healthy rat, which died in 60 hours, with bacilli in all its organs. A bandicot inoculated also showed the bacillus.

The organism is a cocco bacillus, - that is it various in shape, sometimes presenting the round coccal form, at other times the more rod like form of the bacillus. It is small, from 1 to 2 Micro-millimetres in length, and between 0.4 and 0.8 micro-millimetres/

1. Preliminary note on Bacteriological Investigations into the Bubonic Plaque at Bombay Plague at Bombay. by Drs. F. Childe and N.F.Surveyor, Brit. Med. Jour Oct.31 1896 p.1343



Twenty four hours growth of Bacillus Pestis on agar. 1 x 2000 stained with Thionin Carbol.

millimetres in breadth, as seen in its typical form.

The first appearance of the bacillus in Europe was at the Museum of the Pathological section of the British Medical Association at Bristol in 1894. It was shown by Mr. Ernest Hart in specimens sent by Mr. Cantlie from Hong Kong. These specimens were taken from the liver and spleen of a mouse that had been injected with blood from the centre of a femoral bubo. The bacilli were stained with a watery solution of gentian violet, others with an alcoholic glycerin solution of Fuchsin. [1]

It is readily stained by basic aniline dyes, not staining by Gram's method - unless when the spirit used in decolourising is diluted down to 50%, instead of the alcohol being absolute. These cocco-bacilli have rounded ends, and as rule stain more faintly in the centre than at either extremity. The peculiarity is known as "Bipolar" staining, and is very characteristic of the Plague Bacillus.

Mr. Watkins Pitchford considers this staining due to greater condensation of the protoplasm at the ends of the bacilli, giving greater depth of staining. He observes that in unstained specimens of the bacilli this arrangement of the protoplasm can be seen.[2]

1. Brit. Med. Jour. Aug 18. 1894 p.369.
2. Reports on Antiseptics, Bombay April 22nd. 1898 quoted by Balfour Stewart in British Medical Journal, Sept. 23rd. 1899. p.808.

These remarks apply to the appearance of the bacillus in the infected tissues and in the blood. The bacilli in the unstained condition can frequently be seen to be more pigmented at the poles than in the centre. They are, in the tissues, found scattered amongst the cells, either arranged singly, or in twos (diplococcal forms). Occasionally they may be found in chains, (streptococcal forms) particularly in the effusion in the peritoneum of guineapigs and rats.

Involution forms may be met with in the infected organism, particularly in animals which have acquired or which naturally possess a certain amount of immunity to the disease. When obtained from the body some time after death, the bacilli appear swollen and vacuolated, this being evidence of degeneration prior to dissolution in the body fluids.[1] When in this state they lose the property of staining with basic analine dyes.

When stained with ordinary basic dyes the bacillus is occasionally to be seen surrounded by a clear unstained halo. In silver stained specimens, and in those stained by the methods for demonstrating flagella, a definite finely granular faintly stained envelope can be distinctly seen round the bacillus. This probably consists of glutinin.

The/

1. Report of the Indian Plague Commission Ch.III p. 1794.

The plague bacillus is stated to be non-motile.[1] Both Gordon and Kasenky claim to have detected movement occasionally in bacilli obtained from cultures. In a case of Plague Pneumonia in London Dr. (now Sir Patrick) Manson found some motile bacilli in the blood,[2] from the glands of this case typical cocco-bacilli were cultivated. I have myself seen motile organisms in the blood of a patient with a cervical bubo, which I took to be the Bacillus Pestis and certainly considered the movement was more than merely Brownian, although not nearly so active as those of young Typhoid Bacilli. However the mass of evidence is in favour of a non-motile bacillus. Gordon claims to have detected flagella in cultures which have been cultivated for 24 hours on agar at a temperature of 30°C. (86° F).

No spores have been observed although in broth cultivations there are spore like involution forms.

The Bacillus of Plague grows well on the ordinary media, such as agar, gelatin, or in bouillon. If the media be glycerinated it does not grow so well, and it only grows luxuriantly on neutral or slightly alkaline media. During the warm weather in India it is not possible to use gelatin for surface growths, as/

1. Muir & Ritchie. Report of Indian Plague Commission p.55
2. British Medical Journal. Dec 19.1896. p.1794.

as it liquefies. The best growths were found by the Austrian Plague Commission to occur at a temperature of 86° - 97° F., that is, a little below blood heat. Growths can also be obtained at much lower temperatures. Even at the temperature of an ice-safe the Austrian Plague Commission in two series of experiments observed distinct though slow growth of the bacillus, that is , at an average temperature of 44.5° F to 53° F. It cannot grow at a temperature above 107° F.

Oxygen is essential to its luxuriant growth, but even in a tube in which hydrogen has displaced all the oxygen it grows to a slight extent, also in stab cultures in agar or gelatin there is some growth.

The Bacillus is capable of retaining its vitality, and in some cases its virulence, for a long time, as reported by the Austrian Plague Commission; even for several months, if protected from dessicating influences. This may be effected by keeping it at a temperature below blood heat, and by means of placing rubber caps over the mouths of the tubes. If this were not done, it generally died out in seven to eight days at the temperature of blood heat.[1]

The Austrian Plague Commission carried out a series of experiments, most carefully performed, relative to the ability of the bacillus to retain its vitality when cultivated for long periods exclusively on/

1. Report of the Austrian Plague Commission Part II C. p. 630. quoted by Jennings. "A Manual of Plague." p.22

on artificial media, t Out of a series of six cultures which had been grown thus for fourteen months, three were found to have retained their full virulence, two were definitely attenuated, and the third culture gave ambiguous results.[1]

They also found that the virulence was diminished if the cultivations were carried on at blood temperature, a distinct diminution of virulence being noticed after forty three days. When grown at very low temperature 41° - 50° F. it retained its full virulence.

They also chemically altered the media by additions of grape sugar ( up to 5%), glycerin (5%) caustic soda (2%) and hydrochloric acid (0.7%), and found that its virulence is not diminished by growth on media so modified.

The Bacillus can resist great cold. Kasenki reports having found it alive in some of a series of cultures which had been exposed to a temperature of -27° F. but that its virulence was reduced.

Hankin found that the virulence of the bacillus was increased by a series of passages through mice, but that after a series of passages through rats, it was diminished, and this to such an extent that on continuing the passages, the bacillus died out.

This/

1. Report of the Austrian Plague Commission Part II C. p.747. quoted by Jennings. "A Manual of Plague." p.28.

This is a most interesting fact, although the difficulty experienced may have depended on the very considerable resistance to infecting manifested by certain individual rats; but on the other hand it is possible that the virus does become attenuated by passage through each and every rat.[1]

Naked Eye appearances of cultures.

If a sterilised platinum needle is charged with a pure culture of bacilli, either from a culture or an artificial iridium or from the organs or blood of an animal which is infected with bacilli, and stroked along the surface of agar, within fifty eight hours growths appear along the track of the needle. They are at first whitish translucent specks, having a ground glass appearance. They are surrounded, the margins being a little irregular. The colonies increase in size and thickness, the centre presenting a thicker growth than the spreading margins. In time they coalesce and form an irregular colony along the needle track, thick in the centre, thinner at the margins.

On touching this growth with a sterilised needle it is seen to be sticky and adheres to the needle.

In/

1. Report of Indian Plague Commission Ch.III. p.57.

In a growth a few days old the centre becomes yellowish. The surface of the growth is moist. The bacillus grows best in Glycerinised agar. There is no liquefaction of the medium.

On Gelatine the character of the growth is very similar to that in agar and becomes visible on or about the third day. The colonies somewhat resemble the growth of diphtheria, appearing in the form of a white band, increasing in thickness and showing an irregularly knobbed margin. (Klein)[1]

In stab cultures in both agar and gelatine there is only a faint growth along the puncture track which does not increase in thickness, but the growth may spread on the surface of the medium as a whitish film. After some weeks filamentous outgrowths may occur from the stab, more abundant in the upper than the lower part.

1. "A Manual of Plague." Jennings p. 26

Professor Haffkine has laid stress on the growth of the bacillus in bouillon as being very characteristic. A condensed account of this evidence before the Indian Plague Commission is contained in the Report pp.59,60. The main facts are the following :-

On inoculation into bouillon the first appearances of growth occur after a period of 24 hours. Delicate pellicles are found on the surface from the aggregation of floccular masses: these pellicles are first adherent to the sides of the vessel, but later become detached, and on agitation of the tube, sink to the bottom.

Haffkine introduced sterilised olive oil, or cocoanut oil into the culture medium. This oil formed drops on the surface on to which the growths of the bacillus became attached, and from which they grew downwards in the form of tapering stalactites. These, on the slightest agitation of the vessel become detached, and float downwards. As pointed out by the Plague Commission this stalactite growth is conditioned by the following properties of the bacillus -

Firstly, the bacilli adhere together by the glutinous capsule above referred to;

Secondly, the specific gravity of the bacilli being higher than that of the bouillon the growth/

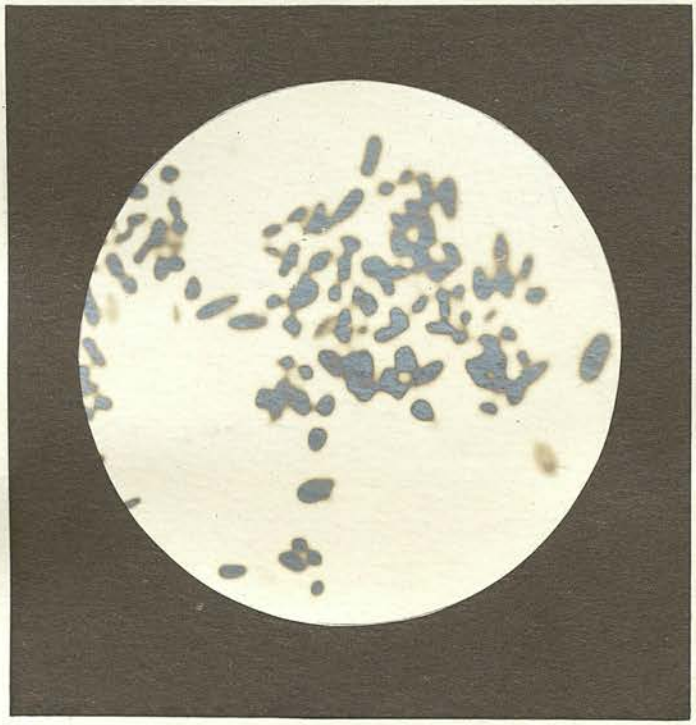
growth is carried downwards;

thirdly, by the fact that the bacteria grow more readily on the surface than below, being in great contact with the oxygen of the air.

Mr. Haffkine reports having met with another bacillus which grows in this way, but the stalactites formed by growths of the plague bacillus are longer, and more easily detached.

Great care must be exercised [1] not to shake the flask accidently, as the stalactites being exceedingly brittle, the least movement, even the careless opening of the door of an incubator in which the preparation may be placed, being sufficient to cause them to drop. Balfour Stewart inoculated his flasks by means of a long thin drawn out glass rod, sterilised in the flame, , the sterilised end being drawn over a culture and inserted to the bottom of the flask. In course of time (he does not state how long) the growth is observed creeping up the glass rod, till it reaches the surface of the broth. It then spreads out, and the surface becomes covered with small round colonies, which from the starting point of stalactite growths downwards./

1. "The Bacteriological Diagnosis of Plague", by C.Balfour Stewart, M.A., M.B. Cantab. British Medical Journal. Sept 23 1899 p.807.



Twenty four hours culture of Bacillus Pestis on salt (3½%) agar. Showing oval and swollen forms. 1 x 2000

downwards.

If the microbe is observed to be growing up the rod in this way, and if the fluid remains clear "it has always turned out to be plague, and shows a stalactite growth in a few days more."
Hankin noticed that when he inoculated broth with plague bacilli isolated from human beings, monkeys and other animals, at the commencement of an epidemic, the stalactite growth was typical; with bacilli isolated towards the end of an epidemic, however, growths were formed which sank to the bottom, and did not form stalactites. This may possibly have been due to a difference between the specific gravities of the broth preparations.[1]

Microscopical appearances of artificially cultivated Bacilli.

Those grown in agar are generally isolated, staining in the typical bipolar fashion. In cultures some days old among typical bacilli are found evolution forms, more usually found in recent cultivations from animals than after several successive growths in artificial media. They are of various shapes, elongated, and branched - spindle shaped, dumb-bell shaped, angular, irregular like amoebae, lemon-or pear-shaped. When freshly cultivated/

1. Report of the Indian Plague Commission. Ch.III p.60.

Twenty four hours broth culture of Bacillus Pestis. Showing chains.

1 x 2000.

cultivated in pure agar they revert to typical forms.[1] If the agar contain from 2½ - 3½% of common salt, exaggerated forms of these bacilli can be produced. This phenomenon is apparently peculiar to the plague bacillus, among other pathogenic organisms, and so is of importance in diagnostic purposes.

Those grown in gelatine are sometimes filamentous in form. These filamentous forms are seen amongst single and diplococcal forms. Klein has demonstrated that only filamentous forms are produced by some gelatine colonies and considers this of importance for diagnostic purposes. [2]

In bouillon cultures streptococcal forms are prone to occur - short chains of six being frequent, but not longer than that.[3]

Bacilli introduced into moist sterilised cow dung retain their vitality for a very long time, and as the floors of native houses are composed of cow dung it is conceivable that the germ finds pabulum in them for its growth and development. Marsh discovered that if agar cultures were buried beneath such floors particularly luxuriant growth occurred in them. He considers this to be due to the large amount of carbonic acid in the ground air.

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1. "A Manual of Plague" Jennings. p. 23, 24.
2. "Report of the Indian Plague Commission Ch.III p.58
3. "A Manual of Plague" Jennings. p. 29.

The bacteriological methods employed in the diagnosis of Plague are -

(1) Microscopical examination of the blood, or other material suspected.

(2) Cultivation Methods.

(3) Inoculation into Animals.

It is recognised by the majority of observers that the bacillus is not observed in the blood until shortly before death . Cantlie[1] speaks of a case of Pestis Minor, which developed the more grave form of plague and in doing so showed a diplococcus in the blood identical with that of Kitasato.

When the bacilli, having the morphological and staining peculiarities of the Bacillus Pestis, are found in considerable numbers in the blood, the diagnosis is certain. There are various outside organisms, diplococcal in form, which much resemble the plague bacillus, these must of course be rigidly excluded. If plague bacilli be not discovered, it is not conclusive evidence that the case is not one of that disease, just as in the case of the blood of a fever patient, the failure to find the protozoa does not absolutely negative a diagnosis of malaria.

If the lymph or pus from a bubo be examined, and the characteristic organisms are seen, especially if/

1. Address before the epidemiological Society. Brit. Med. Jour. Jan.9. 1897. p.72.

if in large numbers , this is "strong presumptive evidence" that the case is one of plague. [1]
Absence of the bacillus, however, does not necessarily negative the diagnosis, especially when the gland is small and may have been missed by the puncturing needle, which may only have penetrated the swollen oedematous tissues round it. In the later stages, again, other organisms may have gained entrance, and supplanted the bacillus of plague.

If the bacilli be found in large numbers in the sputum, the diagnosis is certain.

By cultivation the number of the bacilli may be vastly increased, but the disadvantage of this method is great when there are contaminating organisms present. These inhibit the growth of the plague organism, and so a negative result is of less value when these other organisms are present than that obtained by means of a simple microscopical examination.

Examination microscopically of soil from infected dwellings was found by the Indian Plague Commission to be impracticable, on account of the immense number of organisms which exist in an ordinary native cow dung floor.

An/

1. Report of the Indian Plague Commission Ch.III p.

An isolation method devised by Mr. Hankin consisted in adding to the broth medium a reducing agent and the whole being alkalinised by Ammonia. Although Mr. Hankin claimed to have successfully isolated the bacillus from the floors of infected dwellings by this method, the experiments carried out by the Indian Plague Commission were unsuccessful.[1]

More successful, however, was the method based on Mr. Marsh's observation, that streptococcal forms of the bacillus had grown luxuriantly in cultures buried in the soil, and in those grown in an atmosphere charged with Carbonic Dioxide Gas. The Commission succeeded by this method, in recovering the bacillus from two out of three specimens of unsterilised garden earth which had been infected with a culture of the plague bacillus. These when inoculated into animals caused death, with typical symptoms, and the bacilli were found in the blood of the animals so inoculated.[2]

As regards inoculation of material suspected of being plague-infected into animals, its unsatisfactory nature was demonstrated by a series of inoculations carried out by Mr. Hankin. These were made into mice, the materials being taken from a plague infected room in Bombay, and exceedingly infected it must have/

1. Report of the Indian Plague Commission Ch.III p.64
2. " " " " " " " " " "

have been. Twenty mice first dealt with died from these inoculations. From some of these mice inoculations were carried on through two more series. In all ninety four mice were inoculated, but in not a single instance was the plague bacillus found in the blood or organs,[1] and therefore there is no proof that these mice died of plague.

As is well known to all who have come into contact with native life in India, the methods of floor making employed differ considerably from those adopted in this country.. Their floors are of earth, over which is laid a thin layer of cow dung. This cow dung is applied moist, then it is pressed smooth, and on drying forms a firm level floor. The process of laying down a fresh layer, in cleanly houses, is repeated every few days, each layer being very thin. This cow dung is of course crowded with bacilli of many kinds. In the light of the experiment referred to on page 45 it has been seen that moist sterilised cow dung forms a suitable nidus for the bacillus. Whether it grows equally well in dry unsterilised cow dung is a matter of doubt, as it is not only exposed to desicating influences, but to the inhibiting influences of swarms of other bacilli. If, into the bodies of these animals, the plague bacillus was inoculated amongst other bacilli, it is possible/

1. Report of the Indian Plague Commission. Ch.III p.66

possible that the latter exercised this inhibitory influence upon the organism. On the other hand, previously to inoculation the plague bacilli may have been killed or reduced in virulence, so as not to be capable of inducing an attack of plague.

I have seen no record of experiments performed with the object of ascertaining the length of time required by antagonistic bacteria in order to exert their inhibitory on the growth of the plague bacillus. In the case of the inoculation experiments just referred to, the time that elapsed between the infection of the material, and its inoculation into mice, during which the plague bacilli ( for presumably some were present in the specimens used) were in the close vicinity of, if not in actual contact with antagonistic bacteria, may have been sufficient to so devitalise them as to render them incapable of inducing an attack. Many bacilli which are found in nature outside the body and occur almost everywhere, are known to ne antagonistic to the plague bacillus. Such are the Bacillus subtilis, bacillus mesentericus, the micro coccus prodigiosus, and in a more marked degree, the bacillus coli communis, which letter is found outside the human organism, as well as in the material obtained from it.[1]

SERUM DIAGNOSIS./

1. Report of the Indian Plague Commission. Ch.III p.62

SERUM DIAGNOSIS.

The Indian Plague Commission were of opinion that no practical value attaches to the method of serum diagnosis in cases of plague.

Their reasons for arriving at these conclusions were briefly the following -

(1) As found by the German Plague Commission, negative results were obtained from the serum of plague convalescents.

(2) Not only do plague bacilli naturally mass together into clumps by reason of the glutinous envelope surrounding them, making clumping seen under the microscope of negative value as a means to diagnosis, but these masses when so formed, naturally sink to the bottom of the vessel when detached from the pellicle on the surface, this latter fact compelling us to regard and sedimentation that may be observed as of negative value unless it be especially rapid.[1]

(3) Experiments performed by the commission were very disappointing. They could find no evidence of the presence of a glutinin in the blood of plague convalescents, or of those inoculated with Haffkine's serum or in the antiplague sera from the Pasteur Institute in Paris.

Mode/

1. Report of the Indian Plague Commission. Ch.III. p.68.

Mode of Entrance of the Bacillus into the Organism.

The various modes of entrance of the plague bacillus into the bodies of men and animals is a subject which has received considerable attention, but upon which further light needs to be thrown. It has been proved from accidental inoculations at post mortem examinations, that the virus can be inoculated through slight cuts and abrasions of the skin incurred during the examination. These cuts in some cases have been so slight as to escape observation at the time. The same may also occur through a partially healed scar. At the seat of inoculation there may or may not be local reaction. In the cases where such local reaction has taken place, it has resulted in the formation of a phlyctenule or papule at the site of inoculation. Such have been observed by Captain Childe, of the Indian Medical Service, in the fore arm, calf of the leg, back, and glans penis, and from the phlyctenule or papule in all these cases the plague bacillus was obtained. Fluid from the phlyctaenule in the fore arm and from that on the back was injected into rats, which died of plague.

In other cases the local reaction may be absent or so small as to escape observation.

The theory that infection is most frequently contracted/

contracted through the skin surface, is upheld by this other fact, that in the vast majority of cases the glands affected are those connected with lymphatics originating in the skin. It is very rare for lymphatics of mucous membranes to be primarily involved. In this connection it should be noted that the frequency with which the various glands in connection with the lymphatics are primarily involved varies in direct ratio to the surface of skin represented by those glands. Thus the frequency is greatest in the inguinal glands, than in those of the upper extremity, trunk, neck and head, in order of lesser frequency.1

True it is that the virus can obtain entrance through mucous membranes, such as those of the nose, mouth or pharynx, as is supported by the fact that primary buboes do occur in glands draining from these areas in man, buboes under the chin being of fairly frequent occurrence. It may be introduced through the conjunctiva; that did actually happen in the case of a European nurse in the Parel Hospital, Bombay. A patient coughed up sputum, some of which went into her eye, and she developed an attack of plague, commencing with a bubo just below the ear on the affected side.

As far as our experimental knowledge goes we do not know of any facts which would lead to the supposition that the infection can be conveyed through the stomach or intestine.

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1. Report of the Indian Plague Commission Ch.III p.76.

intestine.

The question of the factors which determine an attack of pneumonic plague is one which demands further consideration. It may be a question of greater virulence of the bacillus, although morphologically it is the same as that causing an ordinary bubonic attack. Or the bacillus may be associated with some other bacterium which favours the production of a plague pneumonia. This plague pneumonia may be the result of a mixed infection, and the great variety of symptoms met with in different cases, would seem to support this view. The German Plague Commission point out in their report[1] that pneumococci may prepare the way for the development of plague bacilli in the lungs.

In 1897 an outbreak of plague occurred in the valley of So-len-ko in Mongolia. The majority of the patients had buboes, but some had none, and most of these latter showed symptoms of pneumonia. One third of the cases had pulmonary symptoms. Dr. Zabolotny observed sixteen cases, seven of which were pneumonic. Four cases he examined bacteriologically and obtained from them cultures of a particularly virulent bacillus.[2]

M./

1. Quoted in Report of Indian Plague Commission. Ch.III. p.73.

2. "Plague in Siberia and Mongolia, and the Tarmagan" by F.G. Clemow, M.D., D.P.H., Journal of Trop. Med. Feb. 1900.

M. le Dr. Batzaroff, of the Bulgarian Army, states that the pneumonic form almost exclusively occurs in the worst epidemics, and includes under this category of pneumonic epidemics, that which occurred under Marcus Aurelius, that of 225 - 265 A.D., the great plague of Justinian 542 A.D., and the Black Death of the 14th Century.[1]

This subject will be further considered when dealing more in detail with the pneumonic form.

With regard to the question of the mode in which the bacillus gains entrance into the bodies of rats some very interesting experiments have been performed by Simond.

It is proved experimentally that it can be inoculated through the skin, but considerable quantities of the bacillus may have to be introduced before an attack of plague be induced ensuring death of the animal. Some rats appear to be remarkably immune.[2]

This latter point was observed by the Austrian plague Commission who stated, however, that in almost all cases inoculation of small quantities was followed by death. The German Plague Commission reported that the prick of an infected needle was sufficient/

1. Annales de l'Institut Pasteur. May 1899 by M.le Dr. Batzaroff, Medicin de l'armée Bulgare.
2. Report of the Indian Plague Commission Ch. III p.74.

sufficient to cause the death of a rat. Corn has been infected with a virulent culture of the bacillus, rats have been fed on this corn and yet have not contracted the disease. When subsequently injected with the same virulent culture they have died in 36 hours. These rats had consumed in all 100 - 150 c.c. of the original plague culture, of which 1 c.c. caused death when injected subcutaneously.

Mr. Hankin conducted experiments also with negative results.

Simond's experiments were briefly as follows [1]

I. A mouse was made to drink a dilution of the Bacillus in water, which, when infected subcutaneously, would kill the animal in 38 hours. Another mouse was fed solely on the same culture on gelatin. Neither mouse showed any symptoms of plague.

II. A culture of the bacillus on gelatin in a Petri's dish was exposed to the air for 48 hours, by the end of which time it was covered with other growths.

One mouse was fed on a dilution of this in water. A second underwent a subcutaneous injection of this dilution. A third was fed on the rest of the contents of the dish.

In none of these mice did symptoms develop.

III./

1. Annales de l'Institut Pasteur. Oct 1898 p.659.

III. One mouse was fed on the liver and heart of a rat which had died of plague.
A second was fed on the body of a mouse dead from experimental plague.
This was repeated in both cases three times within twenty days. They were then inoculated subcutaneously with a culture of plague, and died on the 5th day.
A white and a grey mouse were fed on bread soaked in the blood of a rat which had died of plague. Neither developed any symptoms.

IV. A rat was inoculated with Plague, and then placed in a "bocal de verre" which was provided with a supply of grain. This rat died at the end of three days. A second rat was then put in the same place and made to eat the grain soiled by the first occupant. After five days it was removed, and placed again in the cage from which it had been taken. It developed no symptoms.
A third rat was caused to drink the blood of the one that had died of plague, mixed with a little water. This rat died of plague in four days, but it is important to note that this rat had been wounded in the lower lip, and the cervical bubo which developed showed that infection had been through this and not through the digestive canal.

V. A rat and two mice were fed for two days on pastry with which had been incorporated intestinal contents/

contents of a rat which had died of spontaneous plague. No symptoms developed.

VI. A rat was fed on grain soaked in the sputum of a haemorrhagic case of pneumonic plague. A grey mouse was similarly treated.

A second rat and a second grey mouse drank a dilution in water of the sputum.

None of these contracted plague. The two rats had not been previously encaged, and so may have been immunised against plague.

VII. A rat and a mouse were fed on grain soaked in the urine of a plague patient, obtained seven hours before death. No symptoms.

VIII. A mouse (No.1) was injected subcutaneously with 5 c.c. of fluid obtained by infusing .50 grammes of matter from the intestine of a rat which had died of plague with 5 grammes of water. This mouse died in about 15 hours. No bacilli were found, and no cultures obtained from the blood of the heart.

Mouse No.2 was injected with 1/5 cc of a similar infusion of the intestinal contents of a healthy rat and died in about 15 hours.

Mouse No.3 was pricked with a needle soiled with the dejection of plague rats. No symptoms.

Mouse No.4 was injected subcutaneously with 1/5 c.c. of urine from a plague injected rat. It developed/

developed no symptoms.

Mouse No.5 was inoculated with the blood of the same rat which had died of plague. It died in 49 hours, and was full of bacilli.

IX. A monkey was fed on bread soaked in the sputum of a patient ill with pneumonic plague. No symptoms. The same animal ate bread soaked in the blood of a plague rat and developed no symptoms.

X. A monkey was made to breath plague germs and did not develop symptoms. The same plague infected powder which the monkey breathed was mixed with water and injected into two rats. These rats died in 68 hours.

XI. The foot of a monkey of a very susceptible species was wounded, and the excreta of a plague rat were bandaged over the wound for 10 minutes.

No symptoms developed.

A second monkey of the same species was taken and an excoriation made on the skin surface. Plague sputum first, and then infected soil which had been kept for 24 hours in the dark were brought into contact with the excoriation.

No symptoms developed.

XII. A squirrel was fed on bread soaked in the blood of a rat dead of experimented plague.

No symptoms.

A second squirrel was injected subcutaneously with/

with one drop of the blood of the same rat. This one died of plague after 3 days.

These results differ from those arrived at by the Austrian Plague Commission, who found that an attack of plague is produced in the rat by feeding with the smallest quantity of a growth of the bacillus. They also found that it is only necessary to touch the conjunctiva or intact nasal mucus membrane of a rat with a plague culture in order to induce an attack.[1] Rats are said by them to contract the plague by gnawing the bodies of their companions who have died from it. It does not necessarily follow from this, however, that they contract the disease through the digestive tract, quite possibly the infection gave entrance through the conjunctiva or nasal mucus membrane, through a wound, or possibly from the parasites of the dead rat passing on to the living, carrying the bacillus with them, and inoculating it with their bites.

This leads to the subject of plague in the rat, and the part these animals play in the propagation of the disease.

The fact of rat mortality preceding an outbreak of plague has been noticed above, and is well established. How then does the rat contract the disease?- and how does it communicate it to man?

1. Scheube. "Disease of Warm Climates" p.5

Dr. Simond placed a healthy animal (in three cases a rat and in one a mouse) in a cage, and introduced it into a glass vessel in which was an animal dying of plague. Fleas from a cat were placed in the vessel. One rat and one mouse contracted plague, the other two rats remaining healthy. From this Dr. Simond seeks to show that plague can be conveyed from a sick to a healthy rat by means of suctorial insects. The Indian Plague Commission take exception to his deduction, and show that, owing to the fleas having been taken from a cat and not proved to be parasitic in the rat, it is possible that the infection may have been contracted in some other way.

There is some uncertainty as to whether rat fleas will transmit themselves to men.

Dr. Nuttall has demonstrated that in the case of bugs, plague bacilli are destroyed in their bodies in process of time. He has also proved that no important part is played in the transference of bacterial disease from one animal to another by means of bugs. "Twenty two bugs which had sucked the blood of a mouse dying of plague (the blood contained many bacilli) were immediately placed on four mice. None of the mice sickened after being bitten by the infected bugs."

Healthy rats do not tolerate parasites, when however/

however, they are attacked by disease they lose the necessary energy required in order to perform their toilet, and so the plague stricken rat becomes covered with them. Yersin has discovered that the plague bacillus grows in the intestines of flies which have ingested it. He found that the flies in his laboratory in Hong Kong died in great numbers. He found in their bodies many plague bacilli. He injected a trituration of these into a guineapig which died of plague.[2]

Sablonowski noticed during an epidemic in Mesopotamia in 1884 that a certain fly appeared with the onset of plague and disappeared again. He considered that this fly was responsible for communicating the infection.

The same obtains in the case of fleas, as is almost conclusively shown by Dr. Simond's experiments. He has examined the stomachs of fleas which have been in contact with plague stricken rats, and has found a bacillus morphologically resembling that of plague, although owing to the means at disposal for isolating it from a number of contaminating bacilli being so imperfect, he did not succeed in obtaining a culture. This bacillus, however, was not found in the stomach of fleas which had not been in contact with the plague stricken rats. On inoculating three mice with/

1. Annales de l'Institut Pasteur. Oct 1898 p.674.
2."Tropical Diseases" Manson p.241.

with these fleas, triturated with a little water, one died of plague in 80 hours, the other two died, one after 9 and the other after 12 days. The bacillus, however, was not found in the organs.

He also placed a healthy young rat in the same enclosure as a plague stricken rat with fleas on it. The rats being separated, however, by an iron cage work, thus making contact of the two impossible. On the death of this plague stricken rat he allowed the body to remain for 36 hours, after that length of time removing it. The healthy rat contracted plague, and died on the 5th day.

Then he took the body of a plague stricken rat, robbed it of all its fleas, and let it lie in the same place without fleas with seven healthy rats. Not one of these healthy rats contracted plague.

When a flea bites a rat, it leaves behing it a drop of excreta, which, if it were a culture of the plague bacillus, would be quite capable of causing an attack of plague by penetrating the hole made by the suctorial apparatus. A possibility is that in this way the disease is carried from rat to rat. A plague infected rat dies, the parasites which remained upon it leave the body when death takes place, and seek nutriment on the bodies of healthy animals, communicating the disease to them.

The Plague Commission (p.76 of their Report) consider/

consider that the evidence to hand in support of the theory that plague is carried by fleas from one animal to another is quite inconclusive. No experiments are to hand to prove that the flea can inoculate the bacillus through its dejection into the body of a healthy rat, or that the dejecta contain a living growth of the bacillus.

Giuseppe Zirolia[1] of Rome observed that when a flea has been starved for a time, and then allowed to suck the blood of a plague patient, before sucking, it projects a stream of fluid from its proboscis, without withdrawing the latter. He showed that plague bacilli may remain alive and virulent in fleas for a number of days.

Professor Simpson in Hong Kong inoculated two monkeys with the blood of a rat which had died of plague. They died of the disease on the 6th and 7th days. A third was fed on a banana smeared with the blood of the same rat, and died on plague on the 6th day. A fourth was placed in the same cage with a plague stricken rat. It died on the 10th day.

A cage of three compartments was constructed, the middle compartment preventing all possibility of contact between animals placed one in each end.

A/

1. Jennings. p.41 quoted from Centralblatt fur Bakteriology, Parasteukunde und In Jeklimshrank Neiter. Vol XXXI. No.14 for June 3. 1902

A plague infected rat and a monkey were thus placed. The rat had a great many fleas upon it. It died and on being taken out on the 3rd day it was seen that the fleas had all left the body. The monkey became ill on the 3rd day, presenting all the symptoms of the monkeys that had died in the previous experiments. Eventually, however, it recovered. The inference from this is, of course, that after the fleas left the rat, presumably on or shortly after its death, they became parasitic in the monkey, carrying with them the disease, which they inoculated into it. The monkey may have had a more marked resistance to the action of the virus than the other monkey, or the virus may have become to a certain extent attenuated by passage through the rat, and through the glass, so as not to be lethal to the monkey.

M.M. Wiss and Zabol[1] introduced a culture of the plague bacillus into the trachea of animals under anaesthesia by means of a sound . These animals contracted pneumonic plague. They state that the placing of the virus on the nasal mucous membrane of a susceptible animal, without any excoriation, is followed by pneumonic plague without fail. This has been done on the guinea pig, rabbit and monkey, by means of a fine sterilised rod, with cotton wool in its entremity to prevent wounding.

Small rodents do not always take plague when operated/

1. Annales de l'Institut Pasteur. May 1899.

operated upon in this way, as do monkeys, rabbits, and guinea pigs. But five or six at least out of a dozen, it is stated,will do so. For this purpose a gelatine, not a bouillon growth, is used, as the latter, being light, is expelled by the secretion caused by its introduction.

Only rats recently dead of the disease are capable of conveying it. The reason being, on the above hypothesis, that the fleas all leave the body shortly after death and after their departure the body is harmless. The parasites on leaving the body of the rat wander round it in great numbers over a wide area and attach themselves to other rats, or to human beings. Presumably if these fleas do not find a host they die out, and the bacillus is destroyed. There is no case recorded of the disease being communicated from the handling of a rat after 24 hours from the time of its death. In many cases rats which have died during the day time are removed immediately or those which have died in the night have been handled in the early morning. In these cases great danger lies, and attacks occur in those who have thus handled them. It is now a well recognised fact that on the discovery of dead rats in a house or locality, the only safety for the inhabitants lies in instant flight. After delay of even a few/

few hours in a room in which there is a dead rat, or from which a dead rat has been removed, plague is most likely to supervene.

[1] In Bombay in 1896, a labourer employed in removing rats from a house, died of plague.

One case which I had opportunity of observing was that of my friend and colleague Dr. J. Rutter Williamson, who was attacked by plague in Miraj, S.M.C. in the month of October 1903. It was difficult to account for his being attacked. He was living in a small bungalow on the compound, two other bungalows and some servants' outhouses and go-downs being within the same enclosure, and only separated by thirty or forty yards. No dead rats had been seen in his bungalow, but in the go-down of the neighbouring one, which was at the time unoccupied, dead rats had been found. He had occasion to go into this bungalow for a short time one day, and the possibility is that it was there that the infection was contracted. He had not been in contact with plague patients, or indeed with patients of any kind, previously to his attack, as he was devoting most of his time to language study. The only other channel which was considered and possibility was through the washerman, whose wife died of plague a short time previously. The clothes when/

1. British Medical Journal Jan.23 1897 p.229.

when washed and dried were possibly taken into this man's house, and deposited on the floor near the sick woman. They may have attracted a flea or two with them, which may have been the means of conveying the germs, or the clothes may have acted as carriers of the germ in some other way. The bubo was in the femoral glands.

In the report on the outbreak of Plague in Bombay 1896 - 7 by P.C.H.Snow, Esq., I.C.S. Municipal Commissioner for the city of Bombay, he states that the spread of the plague was due to rats. It did not spread with the fugitives from infected quarters, but with the rats. The Hon'ble Mr. Wadia tells that many dead rats were found in one of his ware-houses. Several people visited the place, but only the coolies who took the rats away died of plague.

It is now quite established by repeated observations that the disease is propagated to man by means of rats and so it may be well to consider what facts are to hand which will throw light upon other modes of infection, and firstly we may consider communication of the disease by

Direct Personal Contact between a plague stricken, and a healthy person, or between the dead body of a plague patient and a healthy person.

A/

A man of the name of Cancelini, who had contracted plague in Marseilles in the year 1720, died six days after returning to his home in Toulon, his decease taking place on October 11th. His daughter died on the 17th of plague. His family consisted of 9 persons, and in less than fifteen days seven members of it succumbed to the disease.

At Wetlianka in Russia, plague was raging. A young girl who had been in this place for three days, returned to her home in Prischib, a small town ten kilometres distant from Wetlianka. She fell ill of plague two days after her return, and died. All the members of her family living with her with the single exception of her grandmother, died within a fortnight. In the same town three sisters of mercy fell ill on the 12th of December, who had returned from Wetlianka on the 8th Inst. They all three succumbed from the 12th to the 15th. A servant died on the 18th; and four residents, who had attended the patients in both this and the foregoing case, and had buried the bodies, died also. No other cases occurred. No one could be found in Prischib to bury the bodies of the latest cases, and two grave diggers were brought from a neighbouring place. These buried them and then were isolated for a month. They remained healthy. The explanation of the immunity/

1. La Semaine Medicale. 1895 p.69. M.le Dr. Netter, Professeur Agrégé à la Faculté de Médicine de Paris.

immunity of these grave diggers may be that they neither attended the plague stricken during life, nor handled them immediately after death, so that before they came near, any parasites that may have been capable of communicating the disease, may have previously left the bodies, which were thus harmless.

We thus see that plague attacks those who have been in close contact with the sick and with dead bodies of plague patients.

In the Egyptian compaign the French Army of 30,000 men lost 1429 of plague and 2419 of other diseases; but among the Medical and administrative staff 260 died of plague and only 49 from other diseases. This fact speaks for itself.

That isolation is a means of protection from plague was proved as long ago as 1605 in Cambridge, in 1720 in Marseilles, at Moscow at the hospice des enfants trouvés in 1771, at the "casernes" and schools of Toura, of Gizeh, of Alexandria in 1835. In Italy the town of Noja, which was strictly isolated in 1815, was alone attacked. At the present time the safety of isolation is proved every year in India and other countries. Europeans who live in bungalows away from the town are practically immune, whilst natives who fly at the approach of plague, and live in temporary houses on the open fields, enjoy freedom from attack.

By/

By several accidents at post mortem examinations it has been proved that plague in inoculable in man from the cadaver.

In 1802 Whyte inoculated himself with bubonic pus by friction. The plague started at the point of inoculation and he died.

In 1835 in the Hospital at Esbekieh there were five condemned criminals. Two of these died after wearing the shirts of plague patients. Three others were inoculated with the watery fluid from a bubo. These got plague.

Dr. Sticker of the German Commission was inoculated in this way a vesicle developing on the right thumb in which plague bacilli were found. Captain Lenmaun, I.M.S. scratched himself at a post mortem on 20th December 1897. On 24th Plague set in. Axillary bubo.

Major Gifford, I.M.S. from a partially healed scar. An axillary bubo developed.

In all thirteen cases are recorded, eleven of these occurring in India.

The infections may also be contracted through the mucous membrane of the nose, mouth, or pharynx. The facts in support of this theory are, that in animals an attack can be produced in this way; that in man buboes under the chin are not at all uncommon; that the plague bacillus has been found in the human subject associated with primary inflammation of tonsils/

tonsils, and mucous membrane of nose and pharynx.

It may get access by the conjunctiva as illustrated in the case of the nurse, in the Parel Hospital, Bombay.

That the infection may be contracted through the mucous membrane of the respiratory passages is not a completely established fact, although it has been so produced in the case of animals, (vide above) and the form has been pneumonic.

The Indian Plague Commission consider it without dount that the clothes and fomites must be infective and retain their infection for a long time.

The poetess[1] brings before us

- the world half blind
'With intellectual light, half brutalised
'With civilisation, having caught the plague
'In silks from Tarsus, shrieking east and west
'Along a thousand railroads, mad with pain -

and cases are ~~in reach~~ *on record* where the infection can only be traced to clothing, bales of cotton, etc.

In Toulon in 1720 a widow left the plague as a heritage with her cast off clothes.[2]

Grassi reports that in 1829 it was carried to the convent of Saint Jean d'Acre after the opening of some cases of clothing which had belonged to persons who had/

1. Eliz. Barrett Browning "Aurora Leigh" Book II. ll.199-203
2. Netter. La Semaine Medicale. 1895. p.69.

had died of the plague two years previously.

Hankin [1] suggests that there is some other means of infection besides the rat, and instances the case of the plague at Hurdwar, a place of pilgrimage near the source of the Ganges. From April 8th to June 8th 1897, 18 cases of plague occurred in a quarter away from the centre of the town. This quarter was evacuated completely, the last case occurred on April 22nd. On May 7th a priest obtained permission to be present at the disinfection of some clothes in the evacuated quarter. He may have slept on these clothes. In any case after seven days he was attacked by plague, and then nine others were attacked in different parts of the town. No rats had been observed to die, and it broke out not amongst grain merchants, but dealers in confectionery. In the course of June rat mortality occurred in the neighbouring town of Kunkhal, near a grain store, but no cases occurred there except that of an old mendicant supposed to have become infected at Hurdwar.

Mead in 1720 speaks of the dangerous nature of plague patients' clothes.

I will instance only a few of the recorded cases in point, which are given in the Report of the Indian Plague Commission.

In July 1897 at Kiamari after a period of quiescence, a case of plague occurred among the Mahrattas. After the opening of a box of clothes which/

1. Annales de l'Institut Pasteur. Oct. 1898.

which had been in the possession of the present owner unopened for three months. The latter was attacked by plague.

A man in Bombay developed fatal plague from using a blanket of his uncle who had died of plague in Lanauli a month before.

Two Goanese stewards, on arrival in London in 1896, contracted plague in a fatal form from wearing clothes which they had brought with them from Bombay and kept in a box until their arrival in England. Several other cases are cited, but these will be sufficient as examples.

It is possible that plague may be communicated through merchandise such as gunny-bags. In the case of Bhujpur the first case was that of a banniah who traded in gunny-bags, which he imported from Bombay. There was plague at Cutch Mandvi, but it was said that he had not been there recently. Three other banniahs were attacked, who probably had trade relations with Cutch Mandvi, so it is possible that the infection was introduced from there.

In the majority of cases plague is imported from an infected to an uninfected place of human communication. In the Punjab, out of 90 villages attacked, 67 were infected by human agency, that is by people who arrived suffering from plague, or who brought infected material with them, but did not themselves/

themselves develop plague. This was the belief of Captains James and Wilkinson I.M.S.[1]

Mr.Mc.Neill believes the same of the Dharwar district and Mr. Stevens, Deputy Plague Commissioner for the Nizam's Territories, of his district. The same holds good for other districts in India.

The question of conveyance by rats cannot be established until human communication between infected and uninfected place can be excluded. Cases are recorded where rats have left villages and wandered into the fields to die, on being attacked by plague, but there is no proof that it is the custom for rats on being attacked, to migrate en masse. It was said that in Bombay the rats all disappeared from the Mandvi quarter of the town, where plague first broke out, and appeared in other parts and all over the city, but the probability seems to be that the rats did not migrate from that quarter, but that they all died, and that when plague spread to those in other parts, they left their holes and came to the surface. So that there is no proof that infected rats migrated spreading plague with them.

Instances are recorded of infection being carried by rats from an infected village to an uninfected village lying in close proximity and in many cases the appearance of dead rats have been noticed prior to the occurrence of the first case of plague. Instance of/

1. Report of the Indian Plague Commission Ch.III p.106.

of this occurred at Bandra, a suburb of Bombay, in Pali Chium, Danda, Santa Cruz, Joo, villages at most two miles apart and some only half a mile.

There are cases of epidemics where no mortality occurred amongst rats. For example, in Poona, where at the time of the first and second epidemics there was no rat mortality in Surat, Nasik, Cutch Mandvi and other places.

Some first epidemics e.g. in Karachi and Belgaum have occurred without rat mortality, the second, however, being accompanied by plague amongst rats. There have, too, been instances of rat plague unaccompanied by the disease in man.

From the course of plague in partially evacuated places it is proved that rats are a means of its spread (p. 125)[2]

Rats are chiefly important in connection with the first outbreak of the disease, but when plague is once established, human agency is of more importance[1]. Koch among many other authorities, holds that plague is primarily an epizöotic, and that the disease in rats is primary to that in men.

Out of 65 villages, the source of infection of which could be traced, 42, or 65% were infected by persons who developed plague after visits to, or residence in, infected villages. In 16 cases(24%) the source was infected articles, in 2 (3%) all infection/

1. Report of the Indian Plague Commission ch.III p.132.
2. Ibid.

infection except that by rats or infected clothes was excluded.

Plague is essentially a disease of filth and overcrowding.[1] When filth and overcrowding do not exist the disease does not spread, where they do, as in so many Oriental rowns, the tendency is to spread. Yet it is an interesting fact that even in the filthiest conditions where overcrowding exists, the disease tends to lose its virulence and to die out after a time. The dwellings of the poorer class of natives in the large cities are of the most insanitary description.

The conditions of life in the country are very much more satisfactory than those in large towns, although even here they are far from ideal. Individual houses are often clean, and as a rule the people perform the natural functions in the fields. But the tanks and wells of the place are as a rule dirty, no hard and fast distinction being made between "drinking" and "washing" water. Cattle are kept in or close to dwelling houses, and there may be heaps of manure close by.

In overcrowding of this sort there is close proximity of the sick and healthy; the floor becomes saturated with excreta; the air of the vilest; vermin abound, both of the body, and others such as rats, mice, etc., which spread the disease. Scarcity of water/

1. "Tropical Diseases." Manson. p.239.

water too, breeds filth of the most appalling description, with neglect of bodily cleanliness, and of that of clothing, food, dishes, and water

Plague occurs periodically in certain houses, and this fact suggests that in all probability the germ can live in the floors of such places as a saprophyte. The free use of disinfectants in some cases has failed to prevent a recrudescence, for the simple reason that there are places in such houses where the disinfectant has not reached, where there is no desiccation and where the germ may have been able to maintain its virulence and growth. In the rainy season the tenants of these places are driven in doors, and the warmth, moisture and carbonic oxide produced by the over-crowding tend to increased virulence of the germ. When the rains commence many rats are driven from their holes and seek refuge in the native houses, become infected, and act as propagators of the disease. 31

Sir Thomas Fraser considers that "the conclusion appears to be completely established that the insanitary conditions existing in the majority of native houses have exerted a direct and important influence, which is indeed a preponderating one, upon the extent and fatality of the present epidemic of plague in India."

The Indian Plague Commission state that "the universal experience of plague in India proves that houses into which the infection of plague has been imported /

imported, whether by men or by rats, are infective, this infectivity being so marked that many of the officers who have had most experience of the disease, have come to the conclusion that the principal source of infection is, as would appear to hold good in the case of yellow fever and possibly also of typhus, to be found in the houses into which the infection of plague has been introduced." Plague is thus said in India to be a "disease of locality." [1]

The contrary conditions ensure almost complete immunity from the disease. Medical man and nurses in well ventilated and clean hospitals rarely acquire the disease.

In Canton and Hong Kong[2] the clean airy European quarters, and the "relatively clean well ventilated boat population were practically exempt, whilst the disease ran riot in the adjoining filthy overcrowded native houses only a few yards away."

Plague appears to attack those in adult life more than the very young or the aged. [3]

The higher rate of incidence among women may be accounted for by their more sedentary mode of life thus being more exposed to infection existing in infected houses,[4] from which the majority of plague cases originate./

1. Report of the Indian Plague Commission Ch. III p.101.
2. Tropical Diseases Manson p.240
3. Report of Indian Plague Commission Ch.III. p.133
4. " " " " " Ch.III. p.138

originate.

[1]Race seems to exert no influence except in so far as it ensures better hygiene. Otherwise what of the great mortality in London in 1665 and of the Black Death of the 14th century?

In this surely lies the great hope for India. She is slow to move, but still she is moving, and Western ideas and practices, though long in establishing themselves, yet are doing so, and perhaps no less surely because slowly. So may we not look forward to the time not only when as a result of a more complete system of irrigation than at present exists, the county will be under more complete cultivation, thus rendering famine a less likely occurrence, but when the people shall have learnt more fully than they have as yet, the habit of living cleanly and healthy lives, in such surroundings as will make it much more difficult, if not impossible for a disease like plague to obtain a firm hold of the population?

Climate does not seem to affect the incidence of the disease beyond influencing it indirectly through modifying the habits of the people.
Epidemics are equally severe in cold and heat, occurring with virulence in the cold of a Russian winter, and the heat of an Indian summer, although as a rule epidemics in Egypt and Mesopotamia declined during the height of a hot and dry summer. [2]

1. A Manual of Plague. Jennings. pp.48-50
2. Tropical Diseases. Manson p.244.

Several observers have remarked that a considerable interval of time is wont to elapse between the introduction of infection into an uninfected district, and an outbreak of plague in that district. Among these are Surgeon-General Bainbridge, Captain James, I.M.S., Dr. Simond and Mr. Hankin. The time that elapses is said to be from three to five weeks. The last observer instances the cases of Kankhal, Satara and Hubli, stating that in each of these places a considerable interval elapsed between the infection of the place and the outbreak of indigenous plague. The Indian Plague Commission consider that the cases of Satara and Hubli are very inconclusive, as in both it was possible that a re-infection had taken place prior to the outbreak of the disease.

Colonel Lawrie, I.M.S. and Mr. Stevens[1] cite an interesting case from Kajurgi, a village in the Hyderabad State, where a banniah arrived from Umarghi, plague infected. He died three days after his arrival. In twenty days time the whole of the bannials family in Kajurgi got plague one after the other, and in all thirteen people died. There had been no sickness during these twenty days, and during that time the infection seems to have become very virulent. No dead rats had apparently been observed in the village.

Dr. Simond seeks from these cases to establish the theory that there is an incubation period, during which/

1. Report of the Indian Plague Commission Ch.III.p.146.

which the disease spreads amongst rats. He instances three cases, in only one of which mention is made of mortality amongst rats in the interval between importation of plague and the outbreak of the subsequent epidemic, and in the other re-infection cannot be absolutely excluded, as the town, Muska, was only three miles distant from the plague-infected Cutch Mandvi. The Plague Commission are of opinion that where there has been a true interval between the importation of the first case and the outbreak of the epidemic, it is bridged over, not by an epidemic amongst rats but by persistence of the bacillus in the soil or in clothes. They also point out the importance of a knowledge of this interval, as until a prolonged period has elapsed after the last case of plague, danger of a further continuance outbreak cannot be said to be past.

As to the occurrence of a-typical forms of plague in the early part of an epidemic there is considerable difference of opinion. Some observers have stated that the first cases are of the mild nature of pestis minor and escape detection as being truw plague. The risk of its propogation being by this mistake increased. The case of Calcutta in 1895 has been instanced. Some cases of malaria, fever, and indolent gland enlargements were notified to the Medical/

1. page 148 of the Report of the Indian Plague Commission.

four
Medical Board, of whom out of six were non-medical men. Five of these were supposed by the medical men who had to deal with them to be cases of pestis ambulans, and one to have been a fatal case of plague. The medical board considered that none of these cases were plague. Some men of the Shropshire Regiment who had been engaged in disinfecting operations in Hong Kong also developed the same symptoms. Dr.Cobb, Consulting Physician to the East Indian Railway, and Dr. W.J.Simpson, Medical Officer of Health of Calcutta, state that they found the bacilli of plague in some of these cases, in the blood.[1]

Surgeon Captain Child, I.M.S. states that he found the bacillus in the blood of patients who had died of "Fever" and of "Bronchitis", in the Sir Jamsetjee Jeejeebhoy Hospital in Bombay.

Epidemics of "mumps" have been noticed to precede the outbreak of the disease. This is stated to have occurred in Poona, and at Kankhal, and in the town of Banga, in the Panjab. This points to a possible infection of the mucous membrane of the nose, throat and pharynx, by an attenuated form of the bacillus.[1] Major Dimmock, I.M.S.[2] instances some cases of acute coryza, with glandular enlargement, and high fever; great swelling of the mucous membrane of the nose and pharynx followed, and death supervened in three or four/

1. Brit. Med. Journal. Feb.27, 1897 p.550.
2. Report of the Indian Plague Commission Ch.III p.149

four days in the severe cases. Professor Haffkine after investigating the cases carefully, could find no evidence in favour of the possibility of their being plague. Later on they became the focus of a severe epidemic of plague in Mazagaon, Bombay.
Only one instance apparently can be brought forward forward of the occurrence of pestis minor preceding the outbreak of severe plague; that is the case of the outbreak in Hyderabad, Sind.
Professor Simpson considers that the outbreak in Calcutta in 1896 was preceded by such mild cases.
The Plague Commission considers the evidence brought forward in support of their view of the early cases inconclusive.[1]

In Bombay during the months preceding the epidemic in 1896 there was a high death rate from respiratory diseases. Captain Childe, I.M.S. there established the fact bacteriologically that plague may assume a pneumonic form. In connection with this allusion is made to the pneumonic form in accounts of the Pali Epidemic in 1836, and the Astrakhan outbreak of 1897 was accompanied as a croupous pneumonia, or as typhus, complicated by pneumonia.[2]

Mr. Whyte, in 1838 mentions the variety with cough, thoracic pain and expectoration of blood, as having occurred first at the various places attacked by plague[3]/

1. Report of Indian Plague Commission Ch.III p.149.
2. Surgeon Captain L.F.Childe, I.M.S. Brit.Med.Journal May 15.1897 p.1215
3. Report of Indian Plague Commission Ch.III p.150

plague. He also notes that the early cases of the plague at Athens were of this nature.

At the commencement of the outbreak in Kumaon and Gurhwal the disease took the septicaemic form.[1]

[2]Various theories have been advanced to explain the interval that may elapse between the disappearance of one epidemic and the outbreak of another in the same place. Such theories have been that possibly cases of pestis minor have occurred during the interval, but it has been shown that the evidence that this condition is contagious is not conclusive; possibly sporadic cases of plague occurring at intervals during the quiescent period keep up the infection. The most likely theory seems to be that the infection is harboured, probably in native houses, between the outbreaks, and on an increase of its virulence, a recrudescence takes place. This theory is supported by various facts - in the first place certain villages have been repeatedly attacked by plague, whilst others have escaped; then in villages where there has been no plague for some years, an outbreak is preceded by a mortality amongst rats; old houses are attacked more frequently than new ones; it is interesting too that women and children are often the first to be attacked, who have not come into contact with outside infection, and/

1. Report of the Indian Plague Commission Ch.III p.150
2. do. do. do. do. do. do. p.176

and who are more exposed, from the nature of their existence, to infection harboured within the houses.

Explanation of the period that may elapse between the introduction of the infection and the outbreak of plague in the place has been sought in the theory that where the germ has been introduced, it requires a certain length of time to become acclimatised to the climatic and other conditions prevailing in that place.

Only when it has become thus acclimatised can it produce a virulent outbreak. On this assumption many epidemiological facts in connection with outbreaks of plague could be satisfactorily explained, such as the slowness of the progress of an epidemic, and its reluctance to spread from the sea coast inland, alternation of periods of activity in infected places with periods of quiescence, on the assumption that the conditions of the environment change with these alternations; the seasonal variations of plague; the fact that although it may spread in one place, in another it may not, although in both places the form it assumes may be equally virulent and fatal, as in the case of Bombay and Calcutta.

Sir Thomas Fraser[1] considers that it is unnecessary to formulate any other theory than that in the houses themselves are found all the conditions favourable for the/

1. Appendix III to the Report of the Indian Plague Commission pp.477,480.

the extension of the disease. He considers that plague has proved itself independent of outside climatic conditions, for within the houses are damp and warmth, calculated to favour its growth, not to mention pollution of the air, which probably also favours it. He considers it proved that the plague bacilli can live for a considerable time in the earth and cow dung floor substance of native houses, despite the presence of contaminating micro-organisms; he also makes mention of experiments performed by Captain Spencer, R.A.M.C. which showed that no obvious deleterious influence was exerted upon the plague bacillus by fluids in which a large number of the organisms found in earth and cow dung had been separately grown.[1]

1. Appendix III of Report of the Indian Plague Commission p.475.

## CLINICAL VARIETIES OF PLAGUE.

The mildest form of plague is that known as Pestis Minor or Pestis Ambulans. Mr. Cantlie calls this type Benign Polyadenitis, in contra-distinction to Malignant Polyadenitis, which latter term he applies to the more severe type of the disease. Cantlie brings forward statistics of a singular type of glandular swellings which occurred in Singapore, the Straits Settlements, and along the coast of China from that region to Hong Kong, between 1892 and 1896.
In 1891, he states that 23 cases were reported in Hong Kong of cervical gland enlargements in children, being enlargement neither of the parotid nor of the submaxillary gland. The condition was epidemic, and accompanied by fever. At that time of course bacteriological proof was not to hand to demonstrate the true nature of these cases, but possibly they were of the same nature as the cases of "mumps" which preceded the outbreaks of plague, above mentioned.

[1]According to Kitasato and Nakagawa it is only an assumption as yet to state that the conditions of enlarged and sometimes tender glands, accompanied by fever for a day or two, which goes by the name of "pestis minor," is in etiological relationship to the/

1. XXth Century Practice of Medicine Vol.XV p.346. Plague. Kitasato and Nakagawa.

the more grave malady. They state their belief that if these cases are really the disease in a mild form, they may exercise a very important influence in its spread.

There are other cases in which the symptoms are prostration, headache, giddiness and gastro-intestinal disturbance, without any apparent glandular symptoms. These cases are observed during plague epidemics, especially in houses where there have been cases of plague, the prostration is great disproportionately to the severity of the symptoms, and this fact, with the additional one that the sequelae of this condition are similar to those of plague proper, tends to show that the one is a mild form of the other.

Cases of pestis minor are very common among those "who have been much exposed to the infection of plague, and are characterised by sensations of numbness and tingling, or by neuralgic pains, which are in many cases associated with the development of shotty glands in the arm pits and the groins." [1] Manson applies the term "Abortive" or "Larval" to a condition of mild plague, and states that the characteristic features are buboes which suppurate or resolve, the associated constitutional symptoms being comparatively mild, or perhaps altogether wanting, although the patient is a long time liable to sudden collapse.[2]

Drs./

1. Report of the Indian Plague Commission Ch.III p.54.
2. Tropical Diseases. Manson p.249.

Drs. Cobb and Simpson have recorded cases of this nature in which they found that a cocco-bacillus was present in the blood and enlarged glands.[1]

Cantlie[2] states that an attack of Benign Roly-adenitis may develop into a malignant attack in the same person, instancing the first case of plague that occurred in Calcutta in 1896. This was that of a boy from Bombay. Fifteen days before leaving Bombay he had noticed swellings in the groins: he never felt ill, however, until his arrival in Calcutta. The Clinical Symptoms of plague then became manifest, and diplococci were found in the blood, identical with Kitasatos.

The incubation period of pestis minor is not known.[3] I have not myself come into contact with these cases of pestis minor, they do not as a rule present themselves at hospital, although cases of recovered plague do appear during an epidemic, frequently, with unhealed indolent ulcers, the remains of buboes which have suppurated. Some of these may have been such cases of Larval plague as Manson describes. These ulcers are very slow in healing.

1. B.M.J. Feb. 27 1897. p.550.
2. B.M.J. Jan. 9 1897. p.72
3. B.M.J. Jan. 9 1897. p.72

I now pass on to notice the characters of the form of plague most frequently met with, namely the BUBONIC FORM. This type is distinguished from the still more severe form, termed "Septicaemic" plague, by the occurrence of primary visible glandular swellings, or buboes.

In the majority of cases the virus gains admittance to the system through a point of inoculation on the skin surface, it then enters the lymph stream, and is carried to the lymphatic glands in relation to the area of skin it has penetrated. On reaching these glands it multiplies, and sets up an acute inflammation resulting in the formation of a bubo which may eventually suppurate. As the greatest area of the skin surface drains its lymphatic stream through the femoral and inguinal glands, and as these glands are in relation to the parts of the body most exposed to infection it is, as would be expected, commonest to find the primary bubo in the femoral and inguinal glands. The same bubo formation occurs in other glands when the virus has gained entrance through other areas of skin, thus there are cases in which buboes are found in the axillary region, in the cervical region, and in the postaural region, according as the infection has taken place through the areas of skin or mucous membrane, or conjunctiva, in relation to the glands in these regions.

Cases/

Cases that are of the bubonic type are not so fatal as those of the septicaemic type, but it must be remembered that one type may pass into the other, a bubonic case developing septicaemic symptoms, or a septicaemic case developing bubo formation. In either group cases may be very mild, or exceedingly virulent.[1]

As regards the incubation period of plague Aozama[2] fixes it between 2 and 7 days.

Hirsch and Summerbrodt at 2½ to 8 days, with an average of 5½ days. The Indian Plague Commission collected a series of 15 cases which allow of the date of infection and the date of onset of the attack being accurately fixed. Of these the incubation periods of five were from 24 to 48 hours, of nine were 3 days, and of one from 3½ to 4 days. Another series of sixteen cases where a history of having come into contact with infection on a particular occasion has been obtained show incubation periods varying from 12 hours to 3 days, ½ of them 4 and 5 days, the shortest period 1 day, being in the case of a woman who on re-entering her vacated house, trod on a dead rat and developed plague the same evening. An instructive incident occurred at Chak Kalal in the Panjab. This village had been evacuated, but a storm of rain drove the villagers again into their houses, where dead rats were/

1. A Manual of Plague. Jennings p.66
2. XXth Century Practice of Med. Vol.XV p.339.

were found; on retiring again to their camps, 29 cases of plague occurred among them. The earliest cases occurred after 48 hours, the largest number on the 3rd and 4th days. From a third series of 41 cases collected by the Commission in which there are histories of the patients having been in contact with infection on and after a particular day the fact is established that plague may develop after an incubation period of possibly 24, certainly 48 hours.[1]

In a certain number of cases there are prodromal symptoms, such as physical and mental depression, feelings of chilliness, giddiness and sometimes dull pains in the groin where the future bubo is about to develop.[2] When present they may last for a few hours only, or for two or three days. Headache may occur during this period, with vertigo, loss of appetite, and nausea. Pain and stiffness in the joints may be noticed.

In some cases this prodromal stage may be absent, or only faintly marked,[3] and the disease sets in more abruptly. On waking in the morning the patient may feel tenderness at the site of the future bubo, in the case of Dr. Rutter Williamson referred to above this was so, and he at first tried to believe that the tenderness in the groin was due to the pressure during the /

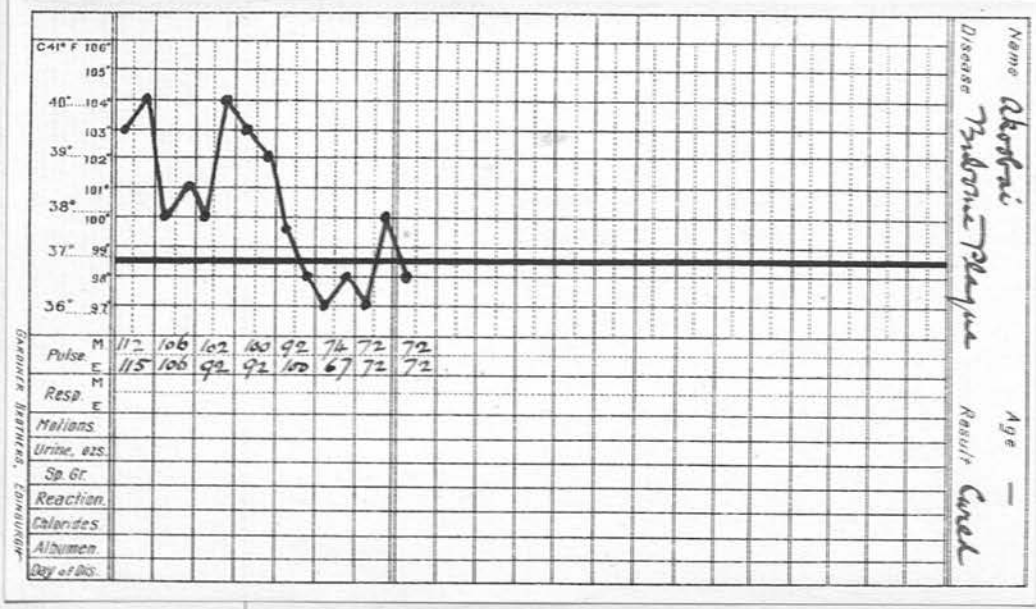
1. Report of the Indian Plague Commission. Ch.III pp.78-86.
2. Tropical Diseases. Manson p.345.
3. Kitasato and Nakagawa XXth Century Practice of Medicine Vol XV. p.339.

the night of the small tassel of his pyjama string. He got up however, and went about his work in hospital without saying anything about it. He looked far from well, tired and somewhat anxious and listless. He fell very ill during breakfast, with faintness, nausea, severe splitting frontal headache and an extremely tender swelling in the femoral glands just below the right groin, the least touch on which was almost unbearable. He had not a distinct rigor, but felt very chilly, although the day was a warm one, and wished for more coverings than usual, and to have the windows of the room closed. He became listless and the temperature began to mount. The face acquired the peculiar expression described by Manson, with the hesitating speech mentioned by Jennings, although at this stage his enunciation was perfectly clear. Severe frontal headache was, I think, the most distressing symptom and kept him from becoming drowsy. His attitude was typical, the right thigh being flexed , to relax tension on the inflamed gland. Mental and physical prostration were very marked, photophobia was also marked, and nausea, accompanied by vomiting. Despite the listlessness the patient was able at this stage to take notice of things going on around, and gave directions concerning various things to be done. The face acquired by degrees the "bloated" expression so characteristic of plague cases.

In/

Right inguinal bubo.

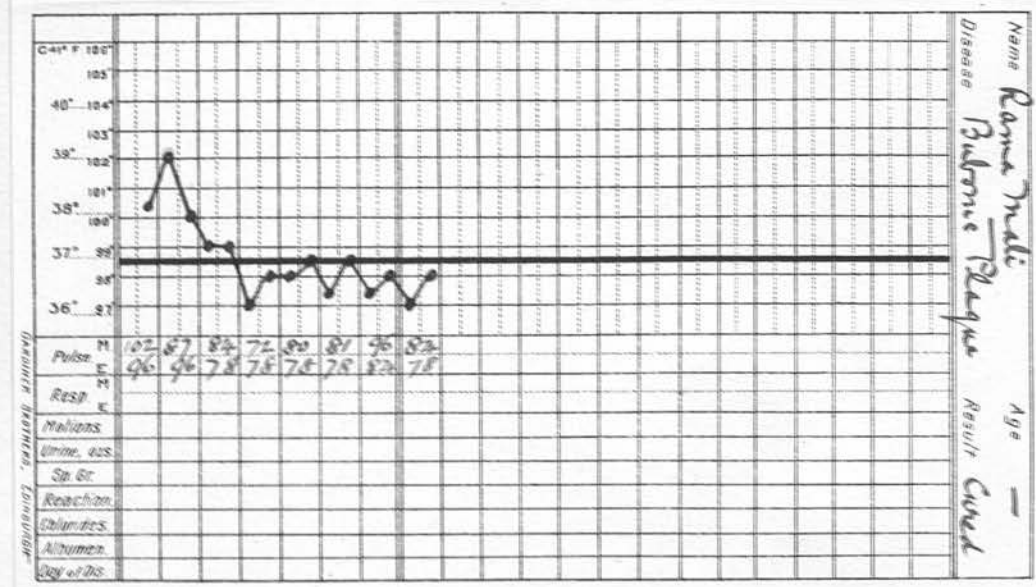
Chart shows a temporary rise after a remission, followed by lysis. The secondary rise possibly being due to suppuration in the bubo.




Right inguinal bubo, the size of a hen's egg.

Fever slight.

Resolution by lysis.



In describing this case I have given an outline of the more important symptoms in this stage of invasion. It may last for a few hours or for several days, before the stage of fever, or fastigium is reached; generally it is of short duration, and may be entirely wanting.[1]

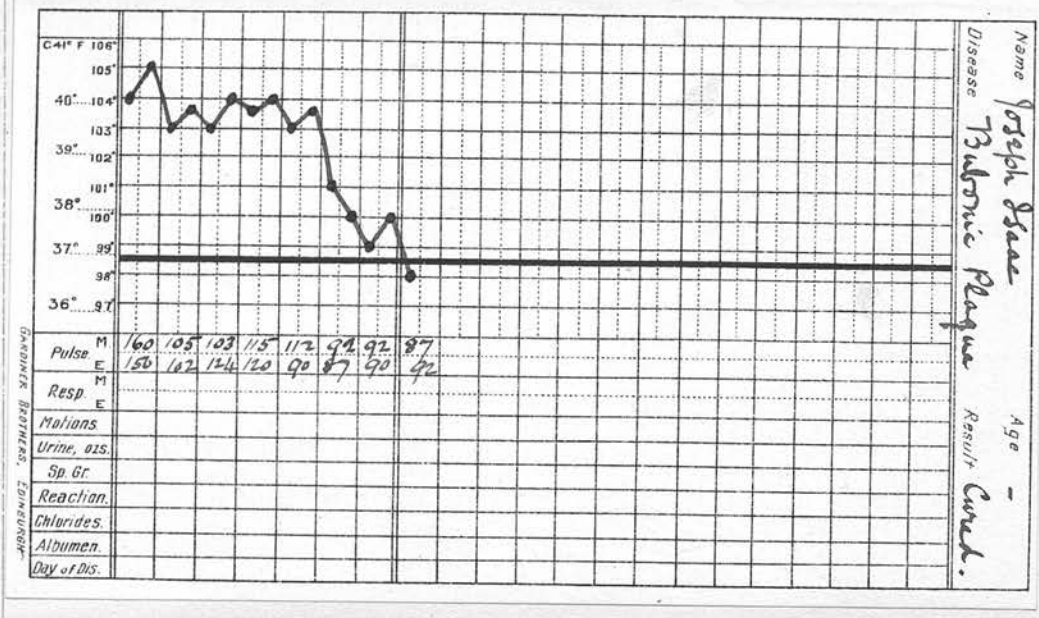
The next stage, that of fever generally commences on the second day. The temperature rises rapidly, from 103° or 104° to a hyperpyrexia of 107° in some cases, with a corresponding acceleration of pulse and temperature. Hyperpyrexia is an unfavourable element in the prognosis. In some cases the rise of temperature may be very gradual, not reaching its maximum till towards the end of the fourth day of the disease. In light cases after fever for three or four days, crisis may occur with profuse perspiration. In more severe cases the fever may continue for a week or more, or even for three weeks. The expression of face becomes more marked; listless, and bloated, the conjunctivae becoming injected and the eyelids swollen. Speech becomes thicker, and the patient so weak that he is hardly able to phonate.

He becomes annoyed on being disturbed, and may show an unwontedly irritable temper. A certain amount of cyanosis may be evident. The pupils may be contracted, normal, or slightly dilated. The patient may breath through his mouth, due to swelling of the nasal mucous membrane/

1. Tropical Diseases. Manson p.245.

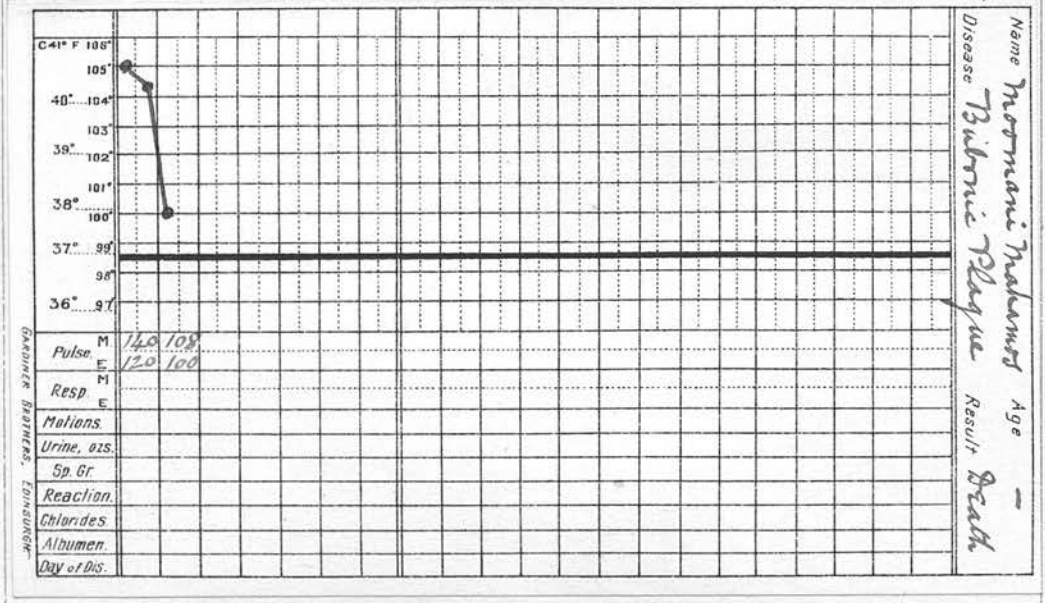
Right cervical bubo. Fever for 36 hours before admission. Showed a higher temperature than the inguinal case, with resolution by lysis.

Case in Miraj Hospital.




Right inguinal bubo.
Great prostration; vomiting, headache, & diarrhoea.
Sudden fall of temperature before death.

Case in Miraj Hospital.



membrane , with consequent obstruction of the nasal duct. The tongue presents in this condition a very characteristic appearance. It is swollen, and indented by the teeth, and covered on the dorsum with a dry, thick, brownish fur, thickest posteriorly. The tip and edges of the tongue are clean and bright red. Sordes form on the teeth, and about the lips and nostrils.

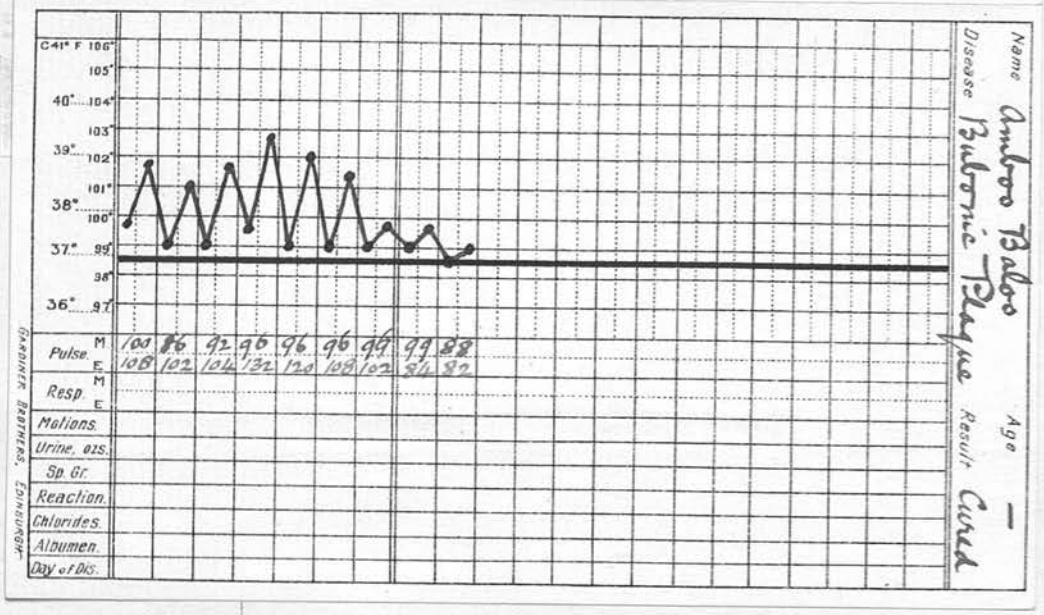
The pulse becomes rapid, more compressible, and dichrotic,and may become intermittent.

Delirium may supervene, either of a muttering type, or with loud shouting,and throwing about of the limbs. This if untreated must tend to heighten the mortality, as the movements may be very violent, and long continued, and a great strain on the already poisoned and weakened vital organs. Sometimes a typhoid stupor sets in. Thirst is extreme. Coma or convulsions, retention of urine, subsultus tendinum, and other nervous symptoms may be observed. Vomiting may occur. Some patients have constipation and others diarrhoea. Urine is scanty, but rarely contains more than a trace of albumin. In the later stages the heart may become dilated, with an enlarged cardiac dulness corresponding, and a weakened or absent first sound.

After the temperature reaches its maximum in very severe cases it may drop by crisis to subnormal, and the/

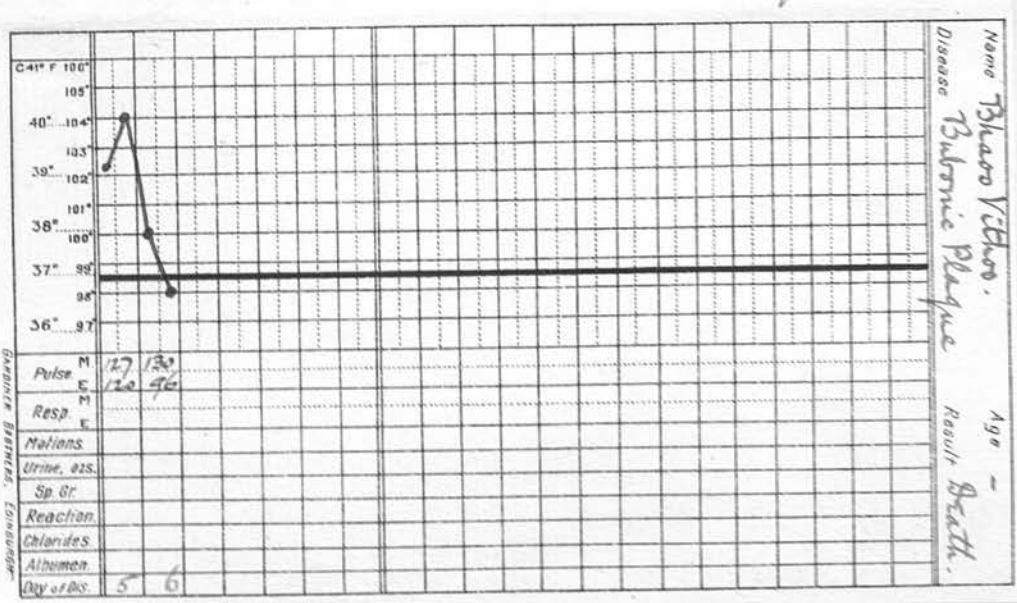
Small right inguinal bubo.
Showing evening rise of temperature, with morning remissions.
Resolution by lysis.

Case in Miraj Hospital.




Left axillary bubo.
Fever for 4 days before admission.
Prostration, diarrhoea, slight delirium.
Sudden fall of temperature before death.

Case in Miraj Hospital.



the patient pass into a condition of collapse; a second or a third rise may however occur before death takes place.

In less severe cases the temperature may show a remission in the morning after the maximum temperature is reached, followed by an evening rise, this occuring for several days until the case terminates favourably by lysis after seven or eight days.[1] If the bacillus gain entrance into the blood stream, of course the symptoms of septicaemia develop, with the characteristic irregular temperature chart of that condition.

When the temperature has gone down convalescence sets in. This is gradually very slow and for weeks and even months the convalescent may suffer from physical and mental prostration. During this time the least bodily or mental exertion causes a feeling of extreme fatigue, even such mild exercise as that of taking a walk along level ground, or of writing a few letters. This may be due possibly to simple cardiac dilation and is doubtless also due in great part to the weakening of the vital nervous centres by the plague poison. If the bubo have suppurated the ulcer may take weeks or months to heal up, and even when healed, at the site of the former swelling, there may continue for a year or two a dragging sensation, reminding one of its having been there. Groin/

1. A Manual of Plague. Jennings. p.72.

Right femoral bubo.
Fever for three days before admission.
Bubo opened spontaneously.
Resolution by lysis.

Case in Miraj Hospital.

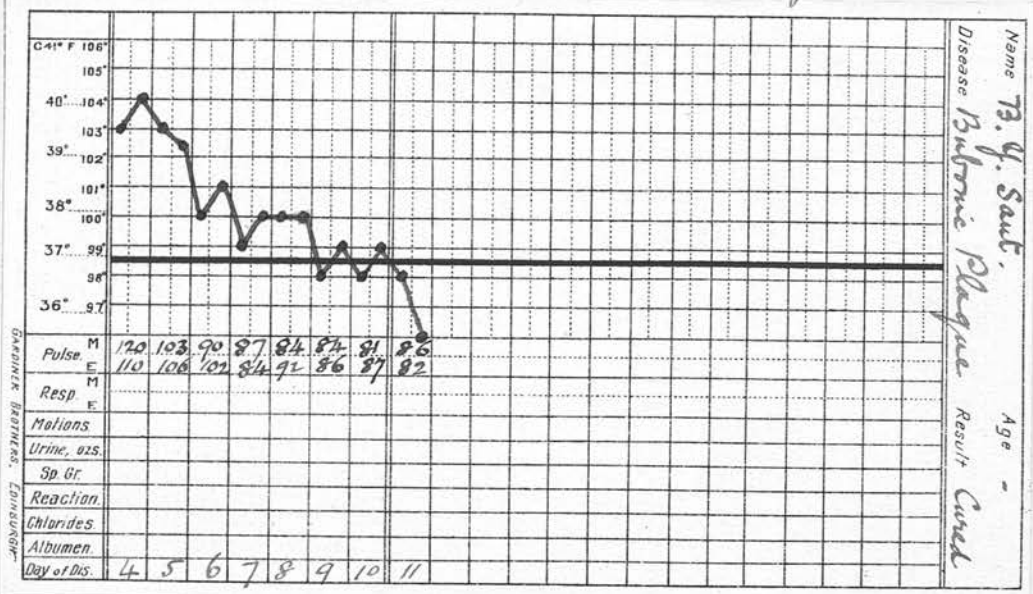
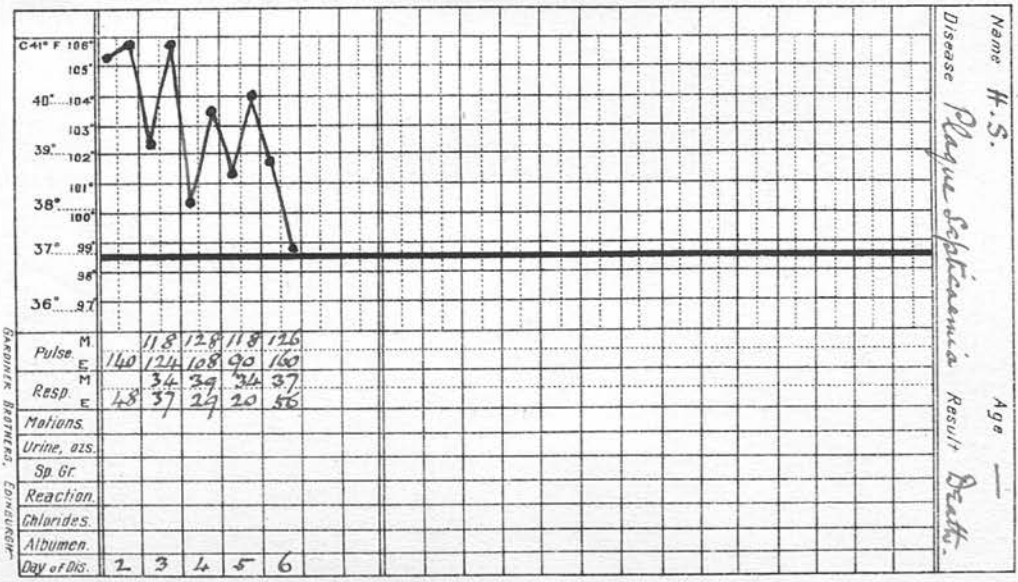



Chart of Plague Septicaemia with multiple superficial buboes, complicated by Secondary plague pneumonia.

Copied from Jennings' "A Manual of Plague". p. 149.



Groin swellings may themselves exist for the length of time, very small, about the size of half a hazel nut on damp days giving rise to mild rheumatic like pains, or feeling of discomfort. Another point noticed is a very sudden coming to the end of ones strength, seeming to indicate a very strong poisoning of the nervous system which has specially affected the cardiac innervation.[1] Various sequelae are of common occurrence to which I will refer later, at this time going over some of the disturbances of the various systems which are characteristic of plague.

As the lymphatic system is the one primarily involved, it may be well to deal with it first. Tenderness, pain, and even swelling of the lymphatic glands may be noticed during the stage of invasions. It is more frequent, however, for these manifestations to be delayed till the period of fever, becoming apparent from a few hours to several days after the first sharp rise, usually on the day following it, the femoral and inguinal glands are most frequently affected, 75% according to Kitasato and Nakagawa occuring in these regions,[2] the swelling being next situated 1 to 1½ inches below Proparts ligament. Next in frequency comes the axillary region 10% The cervical, jugular, submaxillary and suporatyoid, occipital or other groups of glands are more rarely affected primarily.

1. J.Rutter Williamson, M.D., Private correspondence.
2. p.340. XXth Century Practice of Medicine Vol XV. Kitasato and Nakagawa.

I have a case in mind of a woman who for two or three days suffered from swelling and tenderness of the cervical glands, but who did not develop very serious symptoms of plague till the 3rd day of those swellings. She then succumbed rapidly to septicaemic plague, the bacillus being found in the blood a day or so beforehand.

From statistics furnished from three hospitals in Bombay, Karachi and the Panjab, the Plague Commission found that the relative frequency of the occurrence of buboes in the glands of the neck, of the arm pit and of the groin may be expressed by the figures 1: 1.3: 5.8, they also point out the striking similarity between these and the following figures, 1: 1.8: 5, which express the relative area of skin surface draining into the glands of the neck, arm pit, and groin respectively.[1] This tends to uphold the theory that infection occurs through the skin, and that all parts of the skin are equally liable to infection. At the seat of the future bubo there is generally a dull or lancinating pain, with more or less marked tenderness, which increases until it becomes excessive. As the swelling increases the tenderness becomes less marked, the tension in the enlarged gland being relieved by the infiltration/

1. Report of the Indian Plague Commission Ch.III. p.70

infiltration of blood and sero-snaguineous fluid into the surrounding tissues. The swelling may vary in size, it may enlarge rapidly till it becomes the size of a hen's egg or even larger in the course of a couple of days, the size of the bubo depending on the number of adjacent glands affected. A bubo may contain one gland only, or a series of glands, and vary in size and shape accordingly. At first the contour of the glands can be made out to a certain extent, but later on, when there is infiltration into the periglandular tissues, this is impossible, and there is a larger diffuse swelling. When this effusion is very abundant it may lead to oedema and sloughing, from pressure. The bubo may be single, or there may be several. The lymphatic vessels may become affected as well as the glands, and a whole limb may be affected with acute lymphangitis.[1] If the case be a favourable one, resolution of the bubo takes place, but more frequently suppuration. Pus formation does not take place till the eighth or tenth day of the disease, and the majority of severe cases are fatal before the seventh day, so many of the patients die before suppuration sets in. According to Hirsch and Summerbrodt, in light cases, suppuration may occur as early as the third day; it may, on the other hand, be delayed for several weeks. Necrosis/

1. A Manual of Plague. Jennings p.77.

Necrosis may set in, with sloughing of the glandular swelling, in addition to suppuration. The pus and sloughs which are discharged may be very foul smelling.

Cutaneous System. At first the skin is usually hot and dry, and the face flusted.

Cyanosis is not infrequent, but jaundice is rare. Sudamina is rare, but it may occur in the later stages of the disease. Vesicles, pustules, abscesses and carbuncles have been observed. In some there is a slight exanthem, although there is no definite eruption characteristic of plague. Petechiae may occur anywhere but they are most frequent in the affected limb or limbs, and most numerous over the buboes themselves.[1] Extravasations of blood are frequent, and generally most numerous over the buboes themselves; they are often mentioned by early writers, and the name "black death" was given to the disease from their occurrence. They occur less frequently in modern epidemics. Pustules have been known as "plague boils" or "black boils", from the admixture of their contents with blood from capillary haemorrhage. Vesicles have been noted to occur at the seat of inoculation of the virus, when the seat of entrance has been known. This occurred in the case of/

1. A Manual of Plague. Jennings. p.87.

of Dr. Sticken, of the German Plague Commission, over the metacarpo-phalangeal joint of the right thumb. Plague bacilli were recovered from the fluid of this vesicle. A bleb developed in the case of Captain Leumann in which plague bacilli were found.

Captain Childe, I.M.S. instances four cases in which similar local reaction occurred in different parts of the body, associated with attacks of plague. In all of those cases plague bacilli were detected in the vesicles or papules developing locally at the seat of inoculation. Dr. Simond gives 61 cases where there was local reaction at the presumptive site of inoculation, and claims that in every case the bacillus was found locally. In many cases no local reaction is observed, possibly because being slight, it has been overlooked; possibly because it does not exist in the majority of cases, and unless the poison be deposited between the upper and lower layers of the epidermis, a phlyctenule will not develop.

In earlier epidemics the occurrence of cutaneous symptoms seems to have been much more frequent than in modern days.

NERVOUS SYSTEM.

Among the earlier symptoms have already been mentioned headache and vertigo. The patient being very/

very weak, may have the staggering gait of a drunken man.

Cerebral nausea, slowness of thought, irritibility, and restlessness. These symptoms after the period of invasion rapidly becomes worse, passing on to incoordination of muscular movement, persistent and uncontrollable vomiting, lethargy, delirium and convulsions. The delirium is usually much worse at night. Hallucinations may occur, in which he quarrels with his attendants, and the delirium becomes wild; more frequently however, it is low and muttering. Stupor or coma may prevail, or alternate with delirium.

In other cases the mind may remain quite clear throughout the attack, even if it be a severe one Pain in the loins and epigastrium may be complained of[1]. Hiccough, subsultus tendinum may be observed in bad cases. Deafness either the result of general interference with the sensorium, or the result of local disturbances. Stribismus may occur. Stiffness of the neck and clonic convulsions are fairly frequent.

There are generally pains in different parts of the body, as along the course of the nerves; loss of co-ordination of the muscles of/

1. XXth Century Practice of Medicine Vol.XV Kitasato & Nakagawa. p.342.

of speech is very common, giving rise to thickness and hesitancy of speech, weakness of the facial muscles of expression, dulness of hearing, failure of mental power, disinclination to think, muscular tremors, insomnia, impairment of the superficial reflexes, hyperaesthesia and anaesthesia, and local and general muscular spasms.[1]

There is frequently a homicidal or suicidal tendency. Other symptoms named by Jennings are in severe cases jactitation, , carphology, subsultus tendinum, coma-vigil or absolute coma; with, in cases of long standing, evidences of loss of trophic influence such as bedsores, or ulceration of the corneae. The bowels may move involuntarily, but this is rare.

Circulatory System.

At first the pulse is generally frequent, of large amplitude and of low tension, being readily compressible. It becomes rapidly softer, and dichrotic, and if the case be a severe one, intermittent, thready, running, or imperceptible. It is always very rapid, and this rapidity is out of proportion with the increase in the rule of respiration. The extremities are generally cold, due probably to a vaso motor paralysis, to which cause also may be assigned the weakness of the circulation, with the great/

1. A Manual of Plague. Jennings. p.78.

great tendency to syncope. Dilatation of the heart is a frequent occurrence, with production of a systolic murmur at the apex and over the pulmonary area. The apex beat is distinctly visible, and its area is increased; but it becomes weaker, and diastole is lengthened. There is always a danger of heart failure, which may supervene on any undue exertion. It may come on without any warning symptoms, or may be preceded by rapidity and irregularity of the pulse, cyanosis and coldness of the extremities.

In the early stage there may be throbbing in the head and neck, and pain and palpitation of the heart. The blood does not readily coagulate, there is leucocytosis,[1] and the corpuscles do not show much tendency to run into rouleaux.

The poison of plague seems to direct its energies with greatest virulence against the circulatory nervous centres, and it is the heart that above all other organs is weakened in this disease. Treatment as will be seen later, has to be directed towards the upholding of the circulatory power. Haemorrhages into the skin have been mentioned above whilst speaking of cutaneous symptoms.

Respiratory System.

The lungs may be involved either primarily or secondarily/

1. "Almost wholly of Polymorphonuclear leucocytes." Cabot. Clinical Examination of the Blood. p.256.

secondarily. With the primary condition my intention is to deal separately under "Pneumonic Plague." Symptoms referable to the lungs without any particular lesion in them are due to oedema following the congestion which occurs in them as in other organs of the body, such as dyspnoea, rapid respiration, and slight cough, some bronchitis not being uncommon. If pulmonary oedema be very pronounced, or if the cervical glands be much enlarged, dyspnoea may be marked. In a case recorded by Aoyama[1] extreme dyspnoea existed, which at the autopsy was found to have been due to a haemorrhage in the medulla oblongata.

The larynx and trachea are always injected, and there may be haemorrhages into the swollen mucous membrane. There may be oedema of the glottis. Frothy blood stained fluid may be found in the bronchial tubes, the mucous membrane of which is always congested.

Urinary System.

Urine is decreased in quantity, suppression being rare. It may be clear, cloudy, or brownish, and there is usually some albuminuria. A small quantity of blood may be passed, as also may indican. Micturition is frequent. In many cases there is retention, and cystitis may develop. The specific gravity of the urine is high, and reaction acid. Hyaline and epithelial tube casts are observed microscopically.

Urea/

1. Kitasato & Nakagawa. XXth century Practice of Medicine Vol. XV. p.343.

Urea, uric acid and chlorites are diminished. It is difficult to find plague bacilli in the urine, though some observers claim to have done so.

Digestive System.

The tongue is characteristic. A yellowish fur on the dorsum, with clean edges and lips, becoming darker and brownish, peeling off in places leaving raw looking surfaces, the edges and tip becoming later on of a bright red colour. The tongue in the later stages becomes swollen, tremulous, and dry, and indented by the teeth, and the fur a brownish colour. Aoyama does not consider the appearance of the tongue in plague characteristic.

Thirst is extreme, the mouth and fauces dry, sordes covering the gums and teeth. Hiccough is frequently very distressing. Anorexia may occur with nausea and vomiting, the latter in some cases being very persistent; and it does not allay the nausea. The vomited matter is usually watery, but may be bilious, and some blood may be present in it, which does not coagulate. A condition of diarrhoea may set in at first, leading on as a rule to constipation, but diarrhoea may continue throughout the attack, and is an unfavourable complication. The stools are as a rule bilious, and of offensive odour, usually containing blood. Epigastric pain and tymphanites are always present.[1]

The/

1. Jennings. p.86. A Manual of Plague.,

The liver is almost invariably enlarged. Jaundice is rare, and when present is due to a condition of catarrh of the bile ducts or to pressure of the enlarged nodes of liver on the excretory duct.[1] There is tenderness over the hepatic region.
The spleen is almost constantly enlarged by the second or third day, and there is sometimes tenderness over the splenic area.

The tonsils and pharynx are often inflamed and ulcerated, and fatal oedema may supervene on an acute pharyngitis.

Genital System.

In the case of pregnant women it is a very serious complication, as it almost always leads to miscarriage, and death of the mother or child, especially so after the fourth month. A case is recorded in which a mother ill of plague gave birth to an apparently healthy child, the mother dying later of post-partum haemorrhage. The child however, developed buboes in groins and axillae ten hours after its birth, and died eighteen hours later.[2] Plague bacilli were found after its death in spleen and blood, and in the retroperitoneal, femoral, and axillary glands. Infants suckled by plague infected mothers may themselves escape infection. Leumann records 61 instances;/

1. Aoyama quoted by Katasato & Nakagawa. XXth Century. Prac. of Med. Vol XV. p.343

2. A Manual of Plague. Jennings. p.90.

instances; 37 under 18 months old escaped infection, 14 most of them under a month old died of other diseases, the remainder died of plague.

Pneumonic Plague is that form in which the lungs are primarily involved. In animals it has been found that an attack of pneumonic plague can be induced by placing a growth of bacilli on the nasal mucous membrane, or introducing it into the trachea.[1] In December 1896 Childe established that there is a condition of primary plague pneumonia, from bacteriological examination of the sputum. Messieurs Wissokowitz and Zabolotny of the Russian Commission observed several cases of pneumonic plague in Bombay, and produced the disease in the monkey from these cases. They introduced the virus into the trachea of a monkey which was placed under anaesthesia for the purpose. It is stated that[1] on placing the virus on the mucous membrane of a susceptible animal without any excoriation pneumonic plague is produced without fail.[2] This has been performed in the guinea pig, rabbit and monkey by means of a fine sterilised rod, as described above.

The/

1. Annales de l'Institut Pasteur. M.le Dr. Batzaroff- Medicine de l'Armée Bulgare. May 1899.

2. Ibid. p.387.

The symptoms observed are very uniform, in animals operated upon in this way.

During the first 12 hours there is lowering of the temperature to 37. C or lower. This is probably reflex, caused by irritation of the nerves of the mucosa. After this the temperature mounts slowly till the 30th hour, but at this stage plague can only be diagnosed bacteriologically, an examination revealing microbes in process of active proliferation. Following upon this the temperature rapidly mounts to from 40.5° to 44° C, sometimes to 42° C., and remains there. The animal appears distressed, stops eating, its hair stands on end, and respiration is accelerated. The decline is rapid, respiration being more frequent and laboured, it is harsh and noisy and a "veritable roaring" is heard. The sputum is liquid, red stained, and in small quantities. The nasal flow increases, the matter forming yellowish crusts round the nostrils. Conjunctivitis ensues, first on the side of inoculation.

The temperature begins to fall in 24 -36 hours, and the animal dies at the end of the 3rd or the beginning of the 4th day with great dyspnoea and very low temperature. Sometimes paralysis of the stomach and intestine occurs, the swollen abdomen tending to hasten suffocation.

At/

At the post mortem examination secondary buboes are found internally. From time to time primary buboes occur, usually in the neck. Only once was an axillary bubo noticed. On opening the thorax the lungs were found much swollen and did not shrink on the admission of air into the pleural cavity. Haemorrhages of the visceral pleura and infarcts of the lung existed. There were multiple centres of infiltration either separated or massed together so as to occupy the whole of one lobe.

A pure culture was obtained from the blood of the heart.

There was congestion of lung tissue and swelling of the bronchial mucous membrane, and numerous centres of broncho-pneumonia existed. In the alveoli were many bacilli, among desquamated cells and mono-nuclear leucocytes. The pneumonia first produced in those centres is said to have been first a broncho-pneumonia; the virus very quickly pervading the lung and becoming general, the animal succumbs to general septicaemia with oedema of the lungs.

All these conditions were more pronounced on the side of inoculation. The lungs were both oedematous and full of plague microbes. The plague bacilli were also found in the serous fluids. The heart was much dilated, especially on the right side. The spleen was enlarged and of a dark red colour soft and granular on the surface/

surface this being covered with little white spots, this spotted spleen being very characteristic. The stomach was found to be very dilated.

This pneumonic form of plague in animals is infective to other animals, both when the nasal and buccal secretion is placed on the mucous membrane, and by simple co-habitation. In these contacts, absence of buboes and the existence of pulmonary lesions, indicate that the infection was received through the respiratory passages.

Batzaroff believes that there are different kinds of plague bacilli, some of which can exist for months outside the organism, others dying sooner. Some growths which had been exposed to light and air for 3½ months killed animals.

Passage through guinea pigs increased the virulence of the bacillus, and a very attenuated bacillus can be made exceedingly virulent by nasal inoculation through a series of these animals. Some bacilli, he states, may live long when exposed to desiccating influences, and produce pneumonic plague in animals, after 37 days if grown in splenic pulp, and after 19 days if in infusorian soil.

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1. Annales de l'Institut Pasteur. 1898

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1. Annales de l'Institut Pasteur. 1898

of cases the infection had been derived from a previous case of pneumonic plague. Other theories to account for the occurrence of plague pneumonia have been initiated, one is that something specific in the infective material may predispose to the pneumonic form, again that special conditions of form and manner in which the infective material may be communicated from pneumonic patients may exist which determine it; again that it may be accounted for by a mixed infection with pneumococci, streptococci, or other pathogenic bacilli; or that the pneumococci may prepare the lungs for invasion of the plague bacillus.[1]

Primary pneumonic plague is extremely infectious.[2] This might have been expected, when taking into consideration that the bacilli are very abundant in the expectoration, and that they continue to be thrown off from it for considerable periods after the lung symptoms have disappeared, this has been observed even after so long a period as 23 days, although this may have been a case of secondary plague pneumonia. In primary pneumonic plague the bacilli have been observed in the sputum 33 days after the temperature had returned to the normal.

The synptoms of Pneumonic Plague in a characteristic case develop abruptly, and they point to a process of inflammation of the lungs. The respirations become laboured and frequent, cough, pain in the chest, with abundant/

1. Report of Indian Plague Commission Ch.III p.73
2. " " " " " " " p.92

abundant watery frothy sputum which may be tinged with blood in red streaks, but is not the rusty sputum of ordinary croupous pneumonia. There are circumscribed areas of consolidation, and occasionally a whole lobe may be consolidated.

The mass of evidence tends to show that a typical case is of the lobular, not the lobar type of pneumonia. The large majority of the patients die within a few hours of the onset of the disease; in those who survive till the 2nd or 3rd day the involvement of the lymphatic system may be manifested by the development of bubonic swellings in the neck, axilla, or even in the groin.

The physical signs consist of moist and dry rales, which are heard all over the chest, they become crepitant or metallic over the infected parts.

On percussion isolated patches of consolidation are revealed. Dulness may be found over a large area, probably due to the conglomeration of several small patches of consolidation, not to involvement primarily of a whole lobe.

Sir Thomas Fraser writes of the importance this form of plague may assume with regard to the propagation of the disease, and the measures for its control. It was first recognised by Captain Childe, I.M.S. who noticed that in Bombay there was a large increase of death rate besides that due to plague, which had been assigned/

assigned to remittent fever, and disease of the respiratory organs. On further investigation many of those cases were shown to be actually plague. Childe performed a post mortem examination on the body of a patient who had died of this form of the disease. He found by culture methods that plague bacilli existed in the blood and in the spleen. There were pneumonic patches in the first and second stages of pneumonia, and the sputum contained immense numbers of plague bacilli. There were many in the tracheal fluid and a few in the blood and spleen. The left supra trochlea and left inguinal glands showed a fair number, all the other glands very few. This patient had been supposed to be suffering from cough and fever, and there were many others like him.

He quotes the case of a Dr. and his nurse, the latter being attacked by the same form of plague, and dying. The Dr. was attacked in Jan. 2nd dying on the 6th. The nurse became ill of the 7th and died on the 10th. In the sputum of both, plague bacilli were found.[1]

Childe describes what he observed at the necropsy of a Hindu male, aged 25, who died of this form of the disease after 2 days illness. The lungs showed much general engorgement and oedema, with sero-sanguineous frothy fluid in the bronchi, but no pus; the usual appearances of acute bronchitis were absent. There was/

1. British Medical Journal. May 15 1897 p.1215

was one small pneumonic patch, the size of a walnut, in the early second stage, situated below the apex on the front of the right lung, and two similar but smaller patches at the same part of the left lung; these patches stood out a little from the surface and were airless, friable, and sank in water, and each was surrounded by a dark ring of engorgement, which marged into the healthy lung, and there was recent pleurisy over the pneumonic areas. All the other organs were examined and showed considerable engorgement, but no special lesion was observed. The cervical, the axillary, and lumbar lymphatic glands were slightly enlarged, red and soft; all the other glands including the bronchial looked absolutely normal. Microscopically "the pneumonic patches in both stages showed an immense number of plague bacilli, and the rest of the lung showed a large number; the fluid in the trachea also showed very many; the spleen and blood showed a few: the left supra trochlea and the left inguinal glands showed a fair number, and all the other glands extremely few. Cultures in agar-agar were made from the pneumonic lung and spleen, and ultimately a pure growth of the plague bacillus was obtained from each."

Septicaemic Plague.

This term is applied to those cases when from the beginning/

beginning there is a condition of septicaemia or toxaemia, that is where the poison has invaded the whole system through the blood stream directly. These cases are extreme in their severity and in the speed with which they prove fatal, death occuring sometimes a few hours after the onset. It may, however, be delayed till the 2nd, and even till the 5th day in exceptional cases. The term fulminant or lightning plague, or pestis siderans, has been applied to the very rapid cases. The lymphatic glands are involved in these cases, even although in the majority external buboes are not visible. The patient becomes rapidly comatose or delirious, uncontrollable vomiting, haematemesis, haematuria, and malaena follow on, and death is preceded by convulsions. There is prostration from the outset. There may be very little febrile reaction observed.[1] On the other hand the temperature may rise exceedingly high, and in his work on Plague, Major Jennings instances one case in which it rose to 108.4° on the 2nd day followed by a crisis and death, in another the temperature reached 105.8 on the 2nd day.[2]

The septicaemic form may develop from the simple bubonic or from the pneumonic form.

Complications and sequelae of Plague.

Various conditions may complicate the course of a/

1."Tropical Diseases" Manson.p.248.
2. A Manual of Plague. Jennings. pp. 148, 149

a plague attack, among them being subacute rheumatism, arthritis, malaria, phthisis, cholera, erysipelas, relapsing fever, bronchitis, catarrhal or croupous pneumonia, tetanus,[1] gangrene, aphasia, loss of co-ordination of muscles, confusion of memory, etc. Some of the particular manifestations such as suppurating buboes, pulmonary inflammation, petechiae and boils may persist for a long time. Among other sequelae are mentioned peripheral neuritis. Mental impairment, catalepsy, muscular spasm, hemiplegia (partial or complete), pariplegia, facial paralysis, paralysis of the vocal cords, inflammation of the cornea with ulceration and perforation , or destruction of the globe, abscesses and boils, anaemia, dysentery, thrombosis, erysipelas and sloughing.

Pathology and Morbid Anatomy.

The distinctive appearance found post mortem is that of universal dilatation and engorgement of veins and smaller blood vessels, with both minute and large haemorrhages in nearly every part of the body; enlargement of lymphatic glands, which are surrounded by oedema and haemorrhage into the tissues: this generally implicates the external glands, but may involve the glands throughout the entire body. Sir Thomas Fraser considers that the characteristic changes in the glands are largely to be accounted for by changes/

1. Report of Indian Plague Commission Ch.III p.432

changes of the vascular system and that this is responsible in part for the conditions observed in the lungs. He also notes that the changes are very like those observed in cases of such toxaemia caused by the venom of such snakes as the Black Snake (Pseudechis Porphyriacus) of Australia.[1]

I think no useful purpose would be served by going into details with regard to each of the systems. The appearances are all those of engorgement, with congestion of mucous membranes.

As regards the heart, there is generally a condition of fatty degeneration and softening of the muscular fibres. The right ventricles is always dilated, and the other cavities frequently also.

As regards the diagnosis of plague difficulty will chiefly lie with those cases which are very slight and show in only some general glandular enlargement; also those in which external buboes do not appear early, or are absent altogether. In some cases it is impossible to diagnose simply by reference to the symptoms of the individual patient, and account has to be taken of the prevalence or otherwise of plague in the neighbourhood, and the amount of risk of infection that may have been incurred.

The majority of cases where buboes develop are easy of diagnosis, and only at the commencement of an outbreak/

1. Report of Indian Plague Commission. Ch.II p.436.

outbreak might difficulty be experienced.
Bowel complications, such as dysentery, may present difficulties.

In primary plague pneumonia the onset is far more sudden and violent than is the case in ordinary pneumonia and the sputum although it may be tinged with red blood is not of the typically rusty character which is observed in the milder affection.
Much can be learnt from bacteriological examination in pneumonic cases, in the sputum of which bacilli are found in large numbers. In bubonic cases, in the buboes, and in blisters or boils, until suppuration takes place, they are present. In the later stages they may be supplanted by other organisms, such as the streptococci of pus, and so a negative result of examination does not justify a negative diagnosis.

In the blood they are only as a rule found just before death in fatal cases. In the case of blood, cultivation methods are a great advantage, because of greater ease of detection when the bacilli are multiplied in numbers. A positive result obtained by this method will be of more value than that obtained by direct microscopical examination, but a negative result will be less conclusive, because of the increased opportunity given to contaminating micro-organisms to inhibit the growth of the plague bacilli.

Hankine's exaggerated involutions found in agar-agar with $2\frac{1}{2}$ - $3\frac{1}{2}\%$ of salt will help towards a positive/

positive diagnosis, also Haffhine's stalactite growth, and its falling to the bottom on being disturbed, as described by him.

Inoculation of animals is a valuable aid to diagnosis, if the culture of the bacillus be a pure one, otherwise it is unreliable because of the presence of contaminating organisms.

The serum diagnosis is not of any practical value, for the reasons given above.

Prognosis is always grave. The mortality varies in different epidemics and in different periods of the same epidemic. It is generally greatest during the middle period, the cases at the commencement and termination being of a milder type. The pneumonic form is the most fatal, the mortality in some outbreaks being 100%, in others 90%.[1] The Bombay Plague Statistics show a plague mortality (in which all the types of plague are included) of about 75.8%. Sex makes no apparent difference in the mortality except in the case of pregnant women, to whom the disease is very fatal. It is least fatal to very young and very old persons. As regards occupation, in Bombay the greatest mortality was among syces, milkmen, tailors, ayahs, and washermen, although those most affected by plague were mill workers, coolies, and /

1. App.II. of Reports of Indian Plague Commission. p.437.

and domestic servants. Cases with axillary buboes show larger mortality rate than those with buboes in the groin, probably because of the extensive sloughing often caused by them. Patients with buboes of the parotid gland almost invariably die. Cases that are bubonic, even if multiple, are not so fatal as septicaemic cases, but they may become septicaemic, and then the prognosis is of course very much graver. In bubonic cases where the effusion is great the prognosis is graver than in those in which it is slight.

Sanitary conditions affect the mortality to a great extent, especially air and light. Sir Thomas Fraser, in illustration of this, cites the cases of three groups of patients in Bangalore. One was of plague contacts, who, on becoming affected in the well ventilated segregation camp, were removed to the plague hospitals; the second was of plague patients who became affected in the city, and were thereupon removed to the hospitals; and the third group of plague patients who also became affected in the city, but were treated in their own houses. The mortality in the first group was 53.69 per cent, in the second 68.77 per cent, and in the third 97.06 per cent.[1]

In the prognosis of plague it might be taken into account that "nearly all who survive until the eighth day,/

1. App.II. to Report of Indian Plague Commission p. 485.par 85.

day, rather less than four fifths of those who survive until the fifth day, and more than one half of these who survive until the third day, may be expected to recover." [1]

TREATMENT.

The two main lines of treatment are

(1) Prophylactic

(2) Therapeutic . Directed towards cure of those already infected.

Under prophylactic treatment will be included the adoption both of measures calculated to immunise the individual from attacks of plague, and of measures for avoidance of exposure to infection.

For the former vaccines have been prepared by Haffkine and Lustig, and various sera by Lustig, Yersin Roux, Galeotti and others. The latter are applied principally for curative treatment, but they confer a certain amount of immunity, which, although transient is produced immediately. The former do not confer a protective influence till some days after inoculation, although when conferred it is of more lasting duration. Professor Fraser suggests that persons particularly exposed to infection should be afforded immediate protection by the injection of one of the sera, and more lasting protection by subsequent inoculation with/

1. Prof. Sir T.Fraser, App.II. to Report of the Indian Plague Commission. p.442.

with a vaccine. For persons not exposed the latter will suffice.

Conspicuous among those who have worked in connection with the use of vaccines as prophylactics to disease are Pasteur, to whose labours many thousands owe their escape from hydrophobia, and Dr. Ferran who inoculated against cholera but who did not choose to reveal his methods. Haffkine who worked further on the anticholera lines, and thought that the immunity, judging from the somewhat scanty data which could be obtained, appeared to last for a period of 12 months. Yersin, Calmette, and Borrel in 1895 worked at experiments in connection with producing immunity against plague. Haffkine also has done a great deal of work in the preparation and use of anti-plague vaccine.

As the vaccine prepared by Haffkine has been very extensively used in India it will be well to give some account of its preparation. In the first place the difference between a vaccine and a serum is simply that the former consists of living germs and their products, much attenuated (In the case of the anti-rabic vaccine the virus is not killed, but simply attenuated). The latter is the serum of an animal which has been immunised by repeated inoculation of quantities/

quantities of the living germ, first in an attenuated state, then in doses of greater amount and virulence, until at last the animal becomes immune to doses which at first would have proved fatal. Thus the scope of usefulness of a vaccine is in prophylaxis, whilst a serum would be used when the disease has already set in, as a curative agent.

The culture medium used by Mr. Haffkine was obtained by digesting goats meat in the autoclave at 140° C with HCl. and then neutralising the acid medium with Caustic Soda. To ensure proper aeration of the bacilli he introduces oil, which floats on the surface of the medium, the growth attaching itself to the drops and thus remaining near the surface. The medium is inoculated with plague bacilli, and the growths are cultivated at the temperature of the air. If the latter sink below 25°C it is warmed up by lighting gas jets. This process of cultivation is continued for from two to six weeks. The culture is shaken up every few days. Purity is controlled at the close of this operation by inoculating agar tubes, and the growth observed by the naked eye; the character of the growth by which its purity is judged of being the specular appearance of the colonies when seen from the back by reflected light through the thickness of the agar. The next step consists in the killing of the plague bacilli. A sample/

sample is first drawn off with which to control the purity of the culture. The flasks are then placed in a water bath along with a control flask. The bath is filled with water and fitted with a thermometer and the temperature is raised till this thermometer registers 65° C. This temperature is maintained for one hour, at the end of that time to each flask is added a quantity of carbolic acid amounting to one two-hundredth part of the volume of the vaccine. The vaccine was then shaken up, and decanted by syphonage into 4 to 6 ounce narrow-necked, dark - coloured glass bottles, and corked with sterile corks. The sterility of the vaccine was again controlled by inoculating an agar tube with a portion of the first carbolised vaccine syphoned off. The vaccine was then standardised by holding up to the light one or two sample bottles of each brew, and observing the opacity of the fluid. A "Standard dose" being 2½ c.c. the dose of each particular brew was expressed in multiples of a standard dose.

The vaccine consists, when completely prepared, of dead plague bacilli, and of the supernatant fluid, which is simply the nutrient medium in which it has been grown, containing the products of this growth.

As will be seen by all who are familiar with bacteriological methods, there are weak links in the chain/

chain of preparation adopted by Mr. Haffkine. The first point where fault may be found is in this, that the sterility of the vaccine is not tested when proof of it is most urgently required, namely first previous to corking and storing. Secondly the method of standardisation could be improved upon. His method was to observe the effect produced in man by the inoculation of a measured quantity of the vaccine. The standard being the production of an average temperature of 102° F. in a series of 10-20 patients. This is noteworthy, as the vaccine contains considerable quantities of peptone, which is a fever producing element. In the third place his method of standardisation of sample bottles by the amount of opacity he observed when held up to the light is obviously unscientific and liable to error; because for one thing no methods were adopted of ascertaining the virulence of the bacteria, the amount of which was to be judged by the amount of opacity, and for another the quantity could not be accutarely measured in this way. From Dr. Mays' evidence before the Indian Plague Commission it was ascertained that the microbes employed for manufacture of the vaccine were in many cases attenuated by prolonged cultivations in artificial media. It has not yet been proved that non-virulent cultures do not confer any protection, although the German Plague Commission inclined/

inclined to this view. The Indian Plague Commission considered it advisable that the virulence of the bacillus should be kept up by passages through animals.

Measurement of the amount of bacterial sediment by judging of the opacity has lead to serious error, in some cases the weight of the bacterial sediment in a prescribed disease was seven times as large as in others. This variation when it is remembered that the virulence has not been tested, might lead to grave accidents.

The next point to be considered is the relative value of the two main constituents of the vaccine, namely the bacterial sediment of the supernatant fluid.

That the injection of dead bacilli conferred a certain degree of immunity upon rabbits and giunea pigs was demonstrated by Yersin, Roux, Calmette and Borel in 1895.[1] These men believed that no specific toxin is produced by the plague bacillus when it is cultivated in a liquid nutrient medium.[2] The German Commission performed various experiments in monkeys, from which they came to the conclusion that the whole protective power lies in the bacterial sediment. The Indian Plague Commission performed experiments on guinea pigs, and arrived at the /

1. Annales de l'Institut Pasteur 1895.
2. Report of the Indian Plague Commission Ch.IV. p.192

the same conclusion. They consider that the supernatant fluid neither confers immunity against attack, nor lessens mortality; that if there be a specific soluble toxin in the supernatant fluid, it is not one that confers immunity. Very obvious advantages would accrue from dispensing with this supernatant fluid in the performance of inoculations; in the first place the bulk of the injection would be very materially lessened, and with diminished bulk less inconvenience to both patient and operator; in the second place a large quantity of toxic peptone would be eliminated, and less disagreeable result would accrue from the inoculation.

Lustig has also prepared a prophylactic vaccine, and various experiments performed in Berne at the Swiss Serum and Vaccine Institute under Professor Dr. Tavel, Director of the Institute, showed that it is equivalent to Haffkines as an immuniser and has the further advantages that it can be kept longer without deterioration that the reaction is less severe and that the doses can be exactly measured.

It is prepared by inoculating both with a two-days old culture of plague bacilli in serum, in-seminating agar-agar with the broth culture, and adding 1% solution of Caustic potash to the agar agar culture when three or four days old. A slimy mass is thus formed. A 1% solution of Acetic Acid is then added, and a nucleo-proteid from the bodies of the/

the dead bacilli is precipitated. This is dried and pulverised, abd issued for use in quantities of 0.04 grammes dissolved in 21 c.c. of Soda Solution. This is sufficient for three adult doses.

This preparation of Lustig has hot been used to the same extent as that of Hoffkine, so its value has not been so conclusively demonstrated practically.

Mr. Haffkine first observed the physiological effects of inoculation upon himself. He had 10 c.c. of the vaccine injected. The temperature began to rise 3 or 4 hours after the injection and in 8½ hours rose to 102½° F, then began to fall. Tenderness developed at the seat of inoculation reaching up into the left axilla in both flanks, attaining its maximum 15 hours after the time of inoculation. The fever disappeared between 20 and 24 hours after inoculation, the pain lasting for 4 or 5 days at the seat of inoculation, and a nodule remaining for a couple of weeks longer. The same symptoms were observed in my own case in 1902. I found that at the seat of the inoculation there was great tenderness, spreading along the lines of the lymphatic vessels. In my case the amount injected was 5 C.c., only half of that injected in the case of Mr. Haffkine. There was also redness and swelling at the seat of inoculation in the left flank, the redness extending for a short distance along the lymphatic vessels towards the/

the axilla. The vaccine had a fair trial at the Byculla House of Correction in Bombay in January 1897, or rather from the 31st of that month to the 7th of February of the same year. According to the official figures in this case 172 prisoners were not inoculated and 152 were inoculated. Among the non-inoculated there were twelve cases of plague. Six of which proved fatal. Among the inoculated 6 cases of which 3 were fatal. But it would be more just to exclude all cases that occurred after the 4th of February, in so doing making allowance for the elapse of the full incubation period, assuming that to be 5 days. Excluding these cases then,among the inoculated 1 case occurred, which did not prove fatal. From the figure given it does not appear that inoculation performed during the incubation period confers any immunity, as during the first five days after the date of inoculation there were five cases among the inoculated.[1]

The statistics gathered from the whole of India relative to the amount of protection afforded by inoculation with Haffkine's vaccine show great variation. In Byculla jail the ratio which the number of attacks among the uninoculated bore to the number of attacks among the inoculated was as 6.6 to 1. In/

1. Report of the Indian Plague Commission. Ch.IV p.198.

In the case of the hospital at Bangalore it was 1.5 to 1. These are the extreme figures and the ratio at other places varies between them. There are various possible explanations of this variation, the statistics may be sufficiently inaccurate to account for it; under attacks among the inoculated may be included attacks which occurred as the result of infection contracted shortly before or shortly after inoculation; it may be due to difference in the virulence of various epidemics: it may be due to difference in strength and amount of vaccine employed in different places.

It is probable that the first and second of the possible sources of error do cause variation in the ratio; as regards the third possible explanation the facts do not justify us in calculating on variation of the virulence of epidemics being a cause of the difference in the results of inoculation.

Statistics show that some protection is afforded by inoculation during the first three days after the operation, but that it is not nearly so sure as that which ensue after the lapse of the first three days,[1] also that a larger dose confers a greater amount of immunity than a smaller one.

In their summary of conclusions regarding the efficacy of Haffkines vaccine the Indian Plague Commission state them as follows:-

(1) /

1. Report of the Indian Plague Commission Ch.IV. p.258

(1) Inoculation sensibly diminishes the incidence of plague attacks among the inoculated, but the protection afforded is not absolute.

(2) Inoculation diminishes the death rate among the inoculated population.

(3) Inoculation does not appear to confer any great degree of protection within the first few days after the inoculation has been performed, this fact having an important bearing on the risk of infection incurred by recently inoculated persons left in infected surroundings.

(4) The protection lasts for a number of weeks, possibly for a number of months.

(5) The varying strength of the vaccine has had a great effect upon the results obtained from inoculation. There appears to be a definite quantum of vaccinating material, which gives the maximum amount of protection; and provided that this quantum can be injected in one dose, and provided also that the protection turns out to be a lasting one, re-inoculation might with advantage be dispensed with. The best results of inoculation will only be obtained after an accurate method of standardization has been derised.

With regard to the therapeutic value of Yersin's serum/

serum statistical evidence is somewhat slender. Experiments performed upon animals show that it does contain substances which are therapeutically useful, as death can be averted from a plague infected animal by its injection. The amount, however, which had to be injected was very considerable, so that it is doubtful whether much effect could be expected in man from its use. Dr. Simond and Captain Mason R.A.M.C., treated cases with it, and the results of their treatment are such as form presumptive evidence that the course of the disease was favourably influenced by it. Haffkine prepared a serum, but patients treated by it fared worse than those not so treated.[1]

The Indian Plague Commission conducted experiments which did not reveal that the serum aggravated the disease in any way, but the patients did not seem to benefit by the inoculation.

Lustig's Serum was found to apparently diminish the resistance to plague in animals, but hospital records show a diminishment of mortality in cases treated by it, to the extent of 11 per cent in the case mortality of the serum cases as compared with the control cases healed without it.

## MEASURES FOR THE SUPPRESSION OF PLAGUE.

The use of prophylactic vaccine has been already dealt with./

1. Report of the Indian Plague Commission Ch.V. p.319.

with. The other measures that have been resorted to in India include isolation of the infected, segregation of contacts, disinfection of clothing and other articles, disinfection or destruction of houses, according as they are fit or unfit for further human habitation, removal of the inhabitants of infected places to healthy surroundings.

Great opposition against these measures has had to be encountered in various places, and only by degrees are the people becoming alive to their importance.

For the discovery of cases various measures have been carried out. Such as house to house visitation; volunteer agencies for inquiring and reporting; examination of persons coming from infected quarters; rewarding or paying of informers.

The views of the Indian Plague Commission are to the effect that if house searching requires a very strong force to make it effective, as it will never be popular, and the amount of infection must be small; the use of volunteer agencies would lessen the risk of concealment as they come between the people and the executive, and can more easily explain matters to the former's satisfaction.

Surveillance is a useful means of preventing an uninfected place from becoming infected, although it only defers and does not prevent an outbreak, it being next to impossible to prevent people surreptitiously bringing/

bringing themselves and their infected clothing into a healthy part. Rewards and payment for voluntary information have proved useful.

As the earliest cases of an epidemic often escape detection, an outbreak cannot be certainly prevented by these methods.

Compulsory notifications of cases, even if doubtful or suspicious (with punishment for non-notification,) would be difficult to carry out in India, but it would be one means, along with an efficient scheme of death registration,[1] of checking the progress of the disease.

Isolation of the infected can only be useful if the earliest cases can be dealt with; if this has not been possible the best course is to leave the infected place alone, and use disinfectants and inoculation among the other occupants.

Segregation of contacts also, is only of use if done easily, and they should be kept separate until all fear of their communicating the disease shall have passed.

Disinfection of houses is a valuable measure in the case of houses fit for further human habitation. Otherwise complete destruction is the only rational means to employ. In the former case desiccation should also be brought into play by opening up the roofs/

1. A Manual of Plague. Jennings. p.170.

roofs and windows of infected places.

Various experiments have been performed with regard to the value of the various antiseptics. The Indian Plague Commission adopted the method of estimating the value of a disinfectant by its power of destroying all but the more resistant forms of bacteria. Details of their experiments are to be found in the Report.[1] Sterile cotton wool plugs were soiled with the disinfected floor or wall, and small parts of these were placed in nutrient medium, which was of sufficient quantity effectually to dilute any antiseptic that may have been carried over. The tubes of broth were incubated for 24 hours. If growths appeared, isolated cultures were obtained by making zig-zag cultures on agar. Dilutions of disinfectants were prepared, and a sufficient quantity of the bacterium to be examined placed in them. After 15 minutes loopfuls were transferred from these dilutions of disinfectant containing the bacteria to be tested, and broth was inoculated with them, and incubated at the temperature of the room. The resisting power of the various bacilli was then compared with that of the plague bacillus where similarity dealt with by the same disinfectant, and where the resisting power of the latter was found to be distinctly less than that of the other species of micro-organisms from the infected places, the disinfection was assumed to have been adequate/

1. Report of the Indian Plague Commission Ch.VI. p.361.

adequate.

Liston performed experiments under the direction of the Commission showing that although some bacteria resisted disinfection with 1-500 Mercuric Perchloride, and also resisted a 15 minutes exposure to a solution of 1-1000, yet in no case did plague bacilli survive an exposure of the same length to a 1-5000 solution and therefore the disinfection would appear to have been adequate.

"The general consensus of opinion is in favour of mercuric chloride to a 1-1000 solution, which should, inorder to insure complete solution, be made up with Hydrochloric acid or a soluble chloride." [1]

A solution of this strength should be employed, the floors beingsoaked with it, and no corner or crevice being allowed to escape.

Other disinfectants have been employed, such as carbolic acid, phenyl, lysol, permanganate of potash, nitric acid, hydrochloric acid, sulphuric acid, caustic lime, and chloride of lime.

Oxidising agents and acids are shown to be more active in reducing the vitality of the microbe than reducing agents and alkalies.[2]

If desiccation alone be relied upon, ten weeks or more should be allowed to elapse before reinhabitation.[3]

Clothing/

1. A Manual of Plague. Jennings. p.189.
2. A Manual of Plague. Jennings. p.190.
3. Report of the Indian Plague Commission Ch.VI. pp.354-357.

Clothing has been shown to be an important means of spread of plague. Infected clothing should be either boiled in water, exposed to the direct rays of the sun, exposed to saturated steam under pressure, or soaked in disinfectant solutions. Fabrics which would be injured by boiling, such as wool, silk, leather should simply be exposed to the suns direct rays. Furniture should be exposed to the sun, washed with soap and water to remove grease, those with 1-1000 perchloride of mercury. Cushions and other similar things should be destroyed by fire.

The person should be disinfected by bathing in a weak antiseptic solution such as phenyl 1-1000 or carbolic acid 1%.

Evacuation ought to be carried out, when possible, that is, the ppompt removal of all the inhabitants of an infected place to healthy surroundings. This measure largely conduces towards putting down an outbreak. All the property of the evicted should be disinfected,and arrangements made for prompt isolation of cases occurring in the new surroundings, and the people should be prevented from revisiting their houses or carrying infection to other places. This measure is most easily carried out where the earliest cases have been recognised, and where, consequently, only a limited district requires to be evacuated.

Destruction of rats on a large scale is a measure/

measure which should not be neglected.
Devices such as rewards, or employment of professional rat catchers, should be employed. Jennings quotes the opinion of Tsukiyama, the chief of the National Board of Health in Japan, to the effect that it is possible to exterminate the plague infected cities mainly by destroying rats systematically.[1]

He quotes cases of 3 cities where plague had apparently been arrested by this means in"a few months". From such a limited application it is of course impossible to form any valuable opinion as to its efficacy, it seems quite possible that the plague would have died out independently of any such measures, and the total number of rats in the places mentioned cannot have been destroyed, thus leaving others to spread the infection. In a fourth town (Tainan) he states that the plague was brought to an end by this means after it had prevailed for seven years, 80,000 rats being destroyed in this one place in the year 1902.

The Japanese trap rats by means of couple of inverted bowels, so arranged that when the rat nibbles the bait, upon which the smaller bowl rests the other larger bowl, which rests upon the smaller, slips down and imprisons the rat. Poisoning should also be employed by means of arsenic or phosphorus. Virulent cultures of Bac. Typhi Murium have been recommended and used with some success.[2].

1 page 196. A Manual of Plague.
2. Dr. Abel, Annales de l'Institut Pasteur Ap. 1900. Public Health 1902

"The most useful form of trap is a spring-trap baited with ham. Cage traps are too large and the rat soon learns to avoid them." [1]

Measures for prevention of Spread of Plague.

Koch in 1901 stated before the Tuberculosis Congress that doubt no longer existed that in the majority of cases where plague had spread to beyond the sea, that spread had taken place through the intermediary of ship born rats? Manson has insisted upon the necessity for dealing with plague as a rat born disease. [3]

It is pointed out that crews of ships are not those who are attacked by plague; it is rather the dock labourers who handle the cargo.

In view of the fact that in a laden ship it is impossible to kill all the rats, but that this is easy to do in an empty one, it is advisable that all ships should before being loaded, have their rats eradicated.

Most authorities are of the opinion that sulphur dioxide is the best method of accomplishing this. The Belgian council of Hygiene were favourably impressed by the "Clayton Method", in which gas, produced by combustion of sulphur in a special apparatus is driven into the lower parts of the holds of ships, the air being extracted from the upper part till/

1. Public Health 1902. p.370-372.
2. Public Health 1902. p.331
3. Public Health 1902. p.334.

till the air space is permeated with gas to the extent of 10 per cent. One pound of Sulphur is used for every cubic feet of space, 3 per cent of the gas as in the air being fatal to rats.[1]
Dr. Harker, Medical Officer of Health of Tynemouth, and Dr. Ashburn Thompson, of Sydney, both give expression to their belief in the efficacy of Sulphur Dioxide.

At the meeting of the Associated Chambers of Commerce at Nottingham a resolution was proposed by the Bristol Chamber of Commerce, and seconded by that of Greenock to the effect that all captains leaving plague infected ports should be ordered to destroy all rats on their ships before loading cargo, and so take precaution that no rats get on board during loading; and that British consular certificates should be issued to captains obeying these orders.[2]

In the case of an infected ship various regulations were enforced under the Venice Convention which are considered in the Report of the Indian Plague Commission hardly to compensate for the expense and labour involved. The Commission think that all that is necessary in the case of an infected ship is to land and isolate the sick, to disinfect the ship and the effects of those on board, and to permit/

1. Public Health March 1902. pp.368, 369.
2. A Manual of Plague. Jennings. p.207.

permit passengers to be kept under observation at their own homes for a period not exceeding ten days, only those being sent to camps who cannot be kept under observation at home. Only those who cannot be placed under observation in this manner need be placed under detention in a camp.[1]

Measures inaugurated in Bombay in 1896, and adopted there and throughout India generally in order to prevent the spread of plague by railway traffic

(1) All persons travelling from infected places were examined at intervals en route. Any showing undoubted or suspicious symptoms were removed, and detained in plague or observation camp. "Some hundreds of plague cases have been removed annually from trains."[2] Jennings is of the opinion that as practically all persons observed to be suffering from pyrexia, giddiness, suffused conjunctivae, etc., are removed from trains, The probability is that infection is usually conveyed in infected clothes, so that the obvious inference is that the effects of such passengers as are probable carriers of infection should be disinfected.

The disinfection of articles may be carried out by saturated steam under pressure, boiling, soaking in/

1. Report of the Indian Plague Commission ch.VI. p.388.
2. A Manual of Plague. Jennings p.217.

in Chemical disinfectants, or, in the case of articles which will not stand this, exposure to the direct raysof the sun for some hours.

If railway carriages be infected they should be disinfected in the same manner as for dwellings, by Mercuric Chloride.

(2) A system of Surveillance was adopted by which arrivals from infected areas were kept under observation for the period of incubation either at their homes or in camps.

(3) Arrangements were made to disinfect clothing, and passengers'other effects likely to have become infected.

The Indian Plague Commission in their conclusions regarding the efficacy of the various methods employed for prevention of the spread of the disease by railway communication are of the opinion that the benefit accruing from medical examination of passengers has not been large, that any system of land quarantine has not been effective in preventing the spread of plague, and that disinfection of clothing of passengers should not be attempted on the grounds

(1) that if of any value it must be thorough, and a thorough disinfection would cause an amount of delay and inconvenience which would not be justified by the results and

(2) that people can evade the disinfection of their personal/

personal effects by sending them by goods train, the disinfection of acticles sent by goods train being an impossible task.

Measures to prevent the spread of plague by means of communication other than by sea or by railway.

Cordons round infected places may do a certain amount of good. But it is very difficult in India to absolutely prevent passage from one district to another. Merchants have to do so,and native officials may accept bribes, and sometimes go to sleep. Cordons have also been placed round evacuated places in order to prevent people re-entering, but they have proved inadequate;-in the case of that placed round the evacuated railway chawls at Hubli, due to the negligence of the police.

The measures recommended by the Indian Plague Commission may be briefly outlined as follows :- [1]

The methods which have been used in other countries in dealing with infectious diseases in general are these most likely to be of service in dealing with plague, namely notification and isolation of the sick, disinfection of house and personal effects, with, in the case of plague, inoculation as a prophylactic measure.

These/

1. Report of the Indian Plague Commission Ch.VI. pp.401 402.

These measures can only be expected to be imperfectly carried out, so there must always subsist a certain amount of danger.

"A reliable and enormous staff is necessary to carry out measures such as these satisfactorily; and even if funds were unlimited, the staff available would soon prove inadequate in a large county like India."

In places of 2000 to 3000 people (or in the opinion of the President and of Mr. Hewett in places of population up to 10.000 ) the only measures which can be applied are disinfection, inoculation, isolation of the sick, and evacuation of specially infected quarters.

The value of disinfection is especially great when it is applied to imported cases or the first indigenous cases. Segregation of contacts of the general evacuation of large towns have done a great deal of harm, in spreading the disease to uninfected places.

Isolation of the sick can only be satisfactorily carried out by removal to hospital, but in case this is impracticable there is no harm in attempting it at the patient's home. Disinfection is a valuable measure if carried out with thoroughness, although the chances of re-infection by rats or human beings must be borne in mind.

In villages chemical disinfection and inoculation should be resorted to. If many villages become badly/

badly infected the disinfection process should be confined to one or two, and carried out thoroughly in them, and totally adandoned in the others, where evacuation should be relied upon, accompanied by opening up of the roofs and windows of the houses sufficiently to admit sunlight and air. The people should be advised to isolate their sick. The risk of spreading the plague to other places by this measure is probably small where the inhabitants are agriculturalists and their interests accordingly centred in their fields.

It is better to carry out one measure thoroughly than to try to apply all of them with an insufficient and inadequate staff.

In some parts of India the natives evacuate their villages spontaneously on the appearance of plague.

There is no reason why inoculation should not gradually become a more popular measure than it is at present.

The people must depend for protection against plague largely upon their own application of plague measures.

The President was unable to agree with the opinion expressed by the Commission that "chemical disinfection exerted a preponderating influence in destroying the infection where skilled and efficient control was exercised."

He/

He points out in Appendix V to the Report that wherever chemical disinfection has been employed alone, as in Bombay, Parel village, Malabar Hill, Lanauli, Satara, Nasik, Igatpuri, Poona, Ankleshwar, and Jawalapur, "it has never proved sufficient to arrest the progress of the disease," whereas the mere opening up of houses and evacuation without any chemical disinfection, was in several places been successful, as in Sukkur, in many villages in the districts of Dharwar, Surat, Sholapur and elsewhere. Chemical disinfection may however, in his opinion be occasionally useful when used as an auxiliary to sunlight and air, which are the main factors in the process. It is in some cases the only form of disinfection than can be employed, but in these cases it should not alone be trusted to, and prolonged evacuation should also be enforced. It is pointed out too that chemical disinfection does not provide for the difficulty caused by extension of the disease by rats which may reinfect the room as soon as disinfection has been carried out. The President emphasisis the importance of admitting sunlight and air freely into the houses, and considers that this measure is of greater importance than that of chemical disinfection, for one reason that the bacilli are seldom lying free and naked and thus accessible by the disinfecting agent, but are as a rule/

rule enveloped in a covering of sputum and excreta, and on the disinfectant coming into contact with this, a protective envelope would be formed around the bacillus, which would thus remain undestroyed.

The President has enumerated the measures that in his opinion would be employed with advantage at the beginning and at a later stage of an outbreak in large towns, including cities and towns of more than 10,000 inhabitants. These measures include

(1) removal of plague patients to hospital, except in rare cases where isolation in a sufficiently well ventilated and capacious room can be effectively carried out at home;

(2) Isolation of contacts;

(3) Evacuation for at least one month of a considerable area in proximity to, or even surrounding the house or houses in which the first cases had occurred;

(4) Disinfection by chemical substances of the houses and effects of patients.

(5) Disinfection by exposure to sunlight and air of the interior of infected and evacuated houses, wherever required and practicable, and of such articles as cannot without injury be subjected to disinfection by chemical substances;

(6) Wholesale destruction of the rats in the city or town, and if the mice are numerous, of them also:

(7)/

(7) Inoculation with prophylactic fluid of as large a number of the unaffected as possible, with the inducement of shorter residence in the evacuation camps of those who become inoculated.

Provided that the first cases are discovered soon after their occurrence, it is highly probable that the outbreak would be suppressed by these measures.

At a later stage of an outbreak, that is, if the first cases have not been discovered and dealth with, efforts should be made on the lines above indicated, although it is doubtful whether it is possible under these circumstances to suppress the outbreak, although it may be restricted.

In small towns and in villages the President states the following lines of action as being those which if efficiently carried out will with considerable certainty be the means of suppressing the outbreak.

(1) Isolation of patients and of contacts;
(2) Complete evacuation of the town or village for at least one month;
(3) Disinfection by opening up to the influence of sunlight and air of all the evacuated houses.
(4) Disinfection bu chemical substances of the personal/

personal effects of the inhabitants before their removal to the evacuation camp:

(5) Inoculation of as many of the inhabitants as possible.

(6) Destruction of rats, and if mice are numerous, of them also.

"If the first four of the above measures are efficiently carried out it may be confidently anticipated that the outbreak will be suppressed. At the same time, all measures that can be applied for preventing the extension of the disease into other places should also be adopted, and the President thinks that experience has shown that the most effective of them are the regulated supervision of the evacuated, and the securing for them of properly constructed huts in a well arranged and situated camp. [1]

1. Appendix V. to the Report of the Indian Plague Commission pp.509, 510.

Treatment for curing the disease when it has already developed must now be considered.

Good hygienic conditions are very important, such as pure air, absolute quiet, good nursing. Besides these medicinal treatment must be employed and probably helps very materially towards a favourable termination.

The repose of the patient must be complete, and on no account must be allowed to sit up, raise himself on his elbow, or make any exertion. He must remain passive. This must be strictly enforced, not only until the temperature becomes normal, but for at least four days afterwards. The circulatory depression is so great that any such movement is a very serious risk, and may result in sudden cardiac failure and death. I have seen it followed by intense weakness. It is against this circulatory depression that efforts must be made in order to sustain the strength of the patient, this is done by frequent administration of small quantities of liquid food, and by free use of alcoholic and other stimulants, such as aether, camphor, ammonia. This cardiac weakness is an early feature of the disease and so efforts must be made from the onset to combat it. In many hospitals rum is administered immediately after the patient's admission, except in the case of Mussulmans and children. Digitalis and Strophanthus are/

are used to counteract the heart's weakness, and digitalis and strychnine to counteract that of the blood vessels. Strophanthus and digitalis subcutaneously seem to have given the best results, or a combination of digitalis by the mouth with strophanthus subcutaneously Aether is an objectionable remedy when administered subcutaneously as the patient's body and limbs become so sore from its necessarily frequent injection.

Various hypnotics are used to induce sleep and to avoid mental excitement, bodily exertion, and delirium. Morphine may be combined with bromide of potassium or atropine, with cold to the head. Many other hypnotics have been used, such as bromide of potassium and sulphonal. Those should be avoided which particularly depress the circulation.

Elimination should be accelerated by the use of purgatives. Calomel has been found useful by some. Also by the use diaphoretics and diuretics. Tepid sponging has been found a valuable measure for reducing pyrexia. For increasing elimination some have employed a vapour bath at 112° F, followed by partial immersion in water gradually cooled from about 99° to 80° or 75° F, and by friction of the skin.

Injection with one or other of the various antitoxic sera should be performed, a large quantity being injected as early as possible in the disease, and/

and this should be repeated every day for several days. In the septicaemic and pneumonic forms of plague the serum should be injected directly to a vein. The serum should also be injected with the buboes, on the supposition that the virus is at first concentrated in them.

Mercuric chloride and carbolic acid have been administered in large doses, on account of their bactericidal action, but their action is of course not limited to the specific cause of the disease and they combine with many constituents of the body, therefore "fail to exert an appreciable destructive action upon the virus, unless very large, and therefore poisonous doses are administered." 1.

These substances have also been injected into buboes, without any very great advantage accruing from the measure, although suppuration in the buboes is believed to have been hastened.

Local treatment should consist in the application of extract of belladonna and glycerine to the bubo. When suppuration has occurred, but not before there is evidence of it, the swelling should be incised, this incision being followed by the liberal use of antiseptics, such as iodine, mercuric chloride, and lysol.

During convalescence the treatment is dietetic and tonic. Iron, quinine, phosphorus, cod liver oil.

Convalescence/

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1. Prof. Sir T.Fraser Appendix II to the Report of the Indian plague Commission p.444.

2. do. do. do. do. do. p.445.

Convalescence is very slow, the weakness lasting in some cases for many months, cardiac dilation may persist and in the case of those Europeans who can afford it ought to be treated by a course of the Nauheim baths, and resistive exercises, as they are carried out by Dr. Schott. Other treatment for the ensuing cardiac dilation has proved inadequate for its reduction, and so the train of symptoms consequent on it has remained. The ulcers may be exceedingly indolent, the skin necrosis may persist for long, or aphonia or other sequelae before referred to may supervene.

The convalescent should be isolated for at least four weeks after the fever has subsided, especially in pneumonic plague and in cases where secondary pneumonia has supervened.